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MODERN METHODS
IN THE
DIAGNOSIS
OF
PULMONARY TUBERCLE.
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When wilt thou sort an hour great strifes to end?
Or free that soul which wretchedness hath chain'd?
Give phisic to the sick, ease to the pain'd?
The poor, lame, blind, halt, creep, cry out for thee;
But they ne'er meet with opportunity.
The patient dies while the physician sleeps;
The orphan pines while the oppressor feeds;
Justice is feasting while the widow weeps;
Advice is sporting while infection breeds.

(Shakespeare's Lucrece.)

INTRODUCTION.

"Ardua molimur: sed nulla, nisi ardua, virtus".

(Ovid.)

To the modern physician the history of Tuberculosis dates from Koch's discovery and isolation of tubercle bacillus, which he announced to the world in 1882.

The existence of the disease, and also its infectivity have probably been known to mankind, since even previous to the days of Socrates; modern man can no more claim to have discovered the disease than he can to have yet fully worked out its pathology. Apart from this fact however the study of the true pathology of Tuberculosis dates from Koch, and likewise the subject itself, owing to his brilliant work, passes from the blinding mists of speculative pathology into the realms of true science.

When one reflects that whole libraries and not merely single volumes have been written and published on the subject of tuberculosis, it would almost seem a hopeless task, even for a genius in the art of compression, to put into a few sheets of foolscap the main features in the diagnosis of phthisis pulmonalis, consisting in itself alone an exceedingly large branch of the subject. Nevertheless such an attempt is going to be made,

and these main features especially those appealing in any way to the writer constitute the subject matter of this thesis.

The Registrar General whom we must accept as the authority on the subject of "Births and Deaths" informs us that almost one twelfth of the total deaths year by year are due to phthisis pulmonalis.

Facts ascertained from these statistics of the Registrar General, and also from experiment, are now beginning to emerge; paramount amongst these is the one that phthisis pulmonalis is by no means always an incurable disease; in other words the verdict "Consumption" should not, and indeed does not now, always strike terror into the minds of its victims, which it did a few years ago.

Thirty, twenty, even ten years ago laymen and doctors alike regarded the disease with absolute despair. The awful pronouncement that "the lungs were affected" was synonymous with the death warrant of the patient; all that was even attempted in the way of treatment was alleviation of the symptoms. If wealthy, he may have been sent to some warmer climate such as the south of France or the sunny shores of Italy, where under the serene skies and amidst less rigorous climatic conditions, he might linger a little longer perhaps, than in these colder lands of the north; at any rate no degree of hope was held out to him.

Now however "Nous avons changé tout cela";
 "Hopelessness" is becoming "Hopefulness".

I use the word "becoming" guardedly, because unless certain points are carefully and constantly borne in mind, the disease to the unfortunately afflicted is as terrible as ever it was. Towering above every other matter in this respect for the possible future welfare of the individual consumptive, is the importance and necessity of early diagnosis; this also becomes just as important a matter for the general public as it is for the individual. There is probably no other disease where individual and communal interests are so intimate, as in the methods to be adopted when dealing with the tuberculous.

When every woman and child are made to understand that every measure taken for the curing or alleviating of the individual case is equally a measure to prevent their being attacked, then a mighty impulse will be given to the movement now at work to stamp out the scourge. The universal idea that consumption was an incurable disease has probably done more than any other factor has done towards the wide propagation and spread of the disease.

Koch's discovery of the bacillus as I have said was the first fact to throw any real light on the true nature of the disease, and what is even more important it also proved that the disease was a preventible one, in that it was soon after shown

that the bacillus could only successfully attack and overcome those who were first in a low state of health.

When the remark that the disease was a preventible one was made to the late King Edward VII the first words uttered by that shrewd monarch were, "Then why is it not prevented?". At first sight this is a very difficult question to answer, but apart from the yet relatively poor mobilisation of the possible forces at our command, the securing of the early diagnosis of each individual case is probably the most important factor going towards answering this question. In its early stages the disease is not infectious, in its later it is decidedly so; in the early stage it is curable, in the later it is at any rate up to the present incurable.

Nevertheless in spite of all that has been said with regard to the discovery of the tubercle bacillus it is useless and oftentimes positively criminal to withhold a diagnosis from suspected cases of phthisis pulmonalis because the bacillus has not yet been found in the sputum. That this is a practice far from being uncommon cannot be denied, and again and again a positive diagnosis is withheld, and so consequent notification and rational management prevented, because no tubercle bacilli have been found in the sputum of the affected individual.

It is far far easier, and probably on the whole a much wiser proceeding to say a doubtful case is one of tubercle than to say that it is not; it is far far better still however to say a case is one of early phthisis pulmonalis and to know positively that it is so.

The moral is then, that the diagnosis must be made as soon as possible after the bacillus or its toxins begin to overcome the natural resistance of the human body, which is a very different thing indeed from the actual discovery of the bacillus within the suspected patient.

The older school of clinical teachers ^{were} adepts in indicating to us any of the patients up for diagnosis who exhibited signs of the "Tubercular Diathesis" ie those, who though not supposed to be suffering from tuberculosis, were likely to fall victims to it. We were shown at one time the fair-haired, thin skinned, refined and sylph like type of child; or at another the coarse sallow thick lipped, dark haired, scrofulous individual. To my mind there is not the slightest doubt that were those cases before us now and the tuberculin reactions tried one hundred per cent would positively react to a marked degree. In other words the so called 'diathesis' was nothing less than one form of actual tuberculosis, ie: the bacillus was already present within the body; although in spite

of all this, it might be impossible for the most expert of modern clinical pathologists to demonstrate the presence of the actual parasite itself.

These facts become more important still, when we remember that the recent researches of Much and others, have shown that there is a certain group of tubercle bacilli which are not acid-fast and therefore do not show up by the ordinary methods of staining.

Our old teachers may have been wrong when they regarded such types as only predisposed to tubercle; the younger generation have not advanced very much further than they however, in the treatment of such cases.

Keeping to the old pathological classification of cases of phthisis pulmonalis, namely, the primary disposition, secondary consolidation and thirdly excavation; from the treatment point of view, there is no doubt that speaking within broad limits, we may regard all those in the primary stage as curable that many in the second stage may be cured, while all those in the third stage are incurable. From a diagnostic point of view the reverse order holds. The third stage is self evident; it may be difficult to diagnose many cases even in the second stage; it was, until comparatively recently, impossible to diagnose with anything like certainly most of these in the first stage. How important then from a

Treatment point of view does early diagnosis become.

The underlying methods in the diagnosis of phthisis pulmonalis may be classed under four heads as follows:

- I. Symptoms and Physical Signs.
- II. Tuberculin Tests.
- III. Radiography.
- IV. Laboratory Methods.

There is no doubt that the various newer methods now adopted and their comparative value need much further study and elucidation before they can be depended upon by the general medical practitioner; therefore I think no apology is needed for my taking up this subject. It is my intention in this thesis to discuss the various methods in the diagnosis of phthisis pulmonalis and of these more especially those which may be described as the "newer methods"; their relative value and importance will be considered as far as possible the light of my own personal experience. In examining the evidence of the latter it is possible that a benevolent reviewer may recall his Virgil and exclaim "Apparent rari nantes in gurgite vasto", yet fully recognising myself the shortcomings of my task in this respect I should bow to such a verdict and think myself well rewarded.

I must acknowledge my indebtedness to the following gentlemen for the use of clinical material. To Dr. R. Stobo my chief at the

(8.)

Isolation Hospital of the Sunderland Rural District Council for placing at my disposal the Tuberculosis Wards; To Dr. Albert Martin, the Tuberculosis Officer to the Borough of Sunderland for the perusal of his records and charts as well as from time to time the examination of the more interesting cases; To Dr. R.H.Dix and Dr. A.C. Norman for their advice and help on all occasions regarding the place and possible use of radiography in this class of work; To the various residents at the Monkwearmouth and Southwick General Hospital for their invaluable assistance on many occasions I am especially grateful, for which must have been at times rather a monotonous undertaking.

SYMPTOMS.

----- A languor came
 upon him, gentle sickness gradually
 weakening the man, till he could do no more
 but kept the house, his chair, & last his
 bed." (Tennyson's 'Enoch Arden.)

Owing to the immense amount of literature which
 has arisen bearing upon the direct examination
 of the patient, by the comparatively newer methods,
 the importance of this part of the subject has
 probably taken a more backward place than it
 deserves in the estimation of the General Practitioner.

It has been held by some, and I think with
 a good deal of truth that the diagnosis of
 incipient pulmonary tuberculosis can best be made
 by a correct appreciation of the evidence deduced
 from a careful toll of the history and symptoms,
 along with the physical signs observed by the
 examiner; assisted by no other aids than those
 with which nature has endowed every normal
 individual.

Owing to the large amount of what may be
 termed 'Artificial Assistance' the attention given
 to a correct estimate of the symptoms and physical
 signs is not what it ought to be; even if careful
 attention is given, an unduly cautious man is

often afraid of his own judgment, because most of literature, and in many places, also most of the teaching on the subject of Phthisis Pulmonalis deals with tuberculin tests, methods of detecting tubercle bacilli, interpretation of X-Ray photographs, and so ^{on} ~~A~~, probably overlooking the fact, that for the detection of an early case, these means are of little, or even of ~~any~~ value; whatever use they may have in gauging the extent of the lesion and prognosis in later cases.

When a patient comes to consult a medical man he usually comes with some complaint physical or mental, about which some advice is sought. There is probably no disease other than Phthisis Pulmonalis wherein the preliminary statements given by the individuals concerned, are so various and so different.

One may complain of a general feeling of lassitude; another that he has been troubled with a slight cough for some time. Here again a young woman is concerned because her monthly periods have stopped, or ^{have} ~~because~~ become irregular, or another may complain of attacks of cold perspiration at night; then the patient for the first time in the throes of an attack of asthma seeks medical aid, or it may be an attack of haemophysis or acute indigestion, or because he is frequently getting 'colds' in the head and attacks of sneezing, and so on the list may be extended. In all these cases wide differences seem at first sight apparent, yet the underlying

condition - Pulmonary Tuberculosis- may be the same in all. This has to be detected as soon as possible, otherwise cure cannot be looked for.

In all cases a 'history' is most valuable, so a few points concerning this may be considered.

FAMILY HISTORY.

It is not so much with any idea of going into the vexed question of 'Hereditary Tuberculosis' that enquiries into the family history are made. The bearing of hereditary on Tuberculosis is of little practical value and quite beyond the scope of the present thesis.

It is with regard to the environment to which the person concerned may have been exposed, about which the practical man seeks to gather information. What gross infection has this person been exposed to? The word 'gross' is used because, as Sir. A. Newsholme has pointed out " In most instances short exposure to infection does not suffice to infect healthy persons to an extent that will produce serious disease⁽¹⁾", but a healthy person subjected to prolonged exposure to infection as e.g. dwelling in a house or office with tuberculous neighbours, stands very fair chances of becoming infected himself.

It might be argued that these other co-dwellers, even although the presumption is, that they are consumptives, may not be, or have been in an infectious stage at the time. Granted; but the argument that our suspect has not been exposed

to the same infection and surroundings to much the same degree cannot be denied, therefore there exists grounds for this reason alone of looking upon our patient with suspicion. When we come to deal with children of tuberculous parents the case is stronger still, for here we have the naturally greater liability of children to infection along with an even closer environment. Children again need much more individual attention than adults. It is to be presumed, if one or both parents are consumptives, due care and attention will not be given; they must either suffer from this directly on the mother's part, or indirectly through the loss of wage-earning power on the father's. This necessarily results in lowered vitality and therefore greater liability to infection. One may well here quote the conclusions of Halliday Sutherland in his paper on "the Soil and Seed in Tuberculosis"⁽²⁾.

These are as follows;—

(1) There is more tuberculosis among the children of consumptives than among the children of healthy parents.

(2). This may be due to their exposure to infection, to their lowered general health, or to their heredity.

(3). There is more tuberculosis among the children of infectious consumptives than among the children of non-infectious consumptives.

(4). This must be due to exposure to infection, plus lowered general health, insepa-

arable from it as the result of the stage of the disease in the parents.

(5). Therefore it is not heredity which determines whether the children of consumptives will develop the disease, but the existence of certain immediate factors which are under our control.

GENERAL HISTORY.

Much useful information to assist in arriving at a diagnosis can be got by a few pointed enquiries concerning the general history.

What is the occupation?. As is well known certain occupations strongly predispose to phthisis pulmonalis e.g. Clerks, Seamstresses, Filegrinders etc, and all occupations where deficient light and ventilation, overcrowding and long hours are the rule rather than the exception. This does not need dwelling upon.

What is the social position?. Poverty resulting in under-feeding, and poor dwelling houses is one of the most fertile factors.

Previous illnesses?. Most important, Chlorosis Anaemia, and debility following Typhoid, Rheumatic Fever, Syphilis, Chronic Alcoholism, and debauchery generally. Too prolonged a lactation or too frequent confinements; in fact any disease that has resulted in lowered vitality for the individual concerned. The conditions coming under this category are legion and for the most part self-

evident.

It must not be forgotten again that tubercle may have been present in a latent condition from childhood, so enquiries as to health and illnesses at this age period must be made. With such an individual any of the above conditions may act by bringing out the otherwise latent disease.

Sex and Race have their influence, but in the consideration of the individual clinically, these are negligible factors. Age as will be seen lateron, plays a much more important part.

When the present illness is considered and the various symptoms discussed, chronological sequence is not attempted for the reasons already given. What is an early symptom in one may be quite an advanced one in another.

LASSITUDE AND LANGUOR.

Usually pulmonary tuberculosis begins slowly and insidiously, but this all depends on what may be metaphorically described as the virulence of the seed and the fertility of the soil. A feeling of lassitude, languor and listlessness and such vague symptoms may be the first thing complained of, perhaps alongwith loss of appetite and after a little time, also loss of weight. These as stated are only vague symptoms at the most, and so may be common to many diseases, particularly simple Anaemia; yet one must be constantly on the alert especially with one who has recently been exposed to infection, that these symptoms mark not the

onset of tubercle. They may be the outposts of an advance force of the devastating effects in unchecked tuberculosis. This comes forcibly home to me in a case which came to my notice during my first year of practice shortly after qualifying.

A young woman, married, 24 years of age complained of being generally out of sorts along with loss of appetite. There was no cough. She had had amenorrhoea for two months. A simple anaemia was diagnosed along with the possibility of pregnancy. She was put on an iron and arsenic mixture and received in all two eight ounces bottles from me. I saw no more of her. Her subsequent history I got from a colleague; he told me that he had been hurriedly called to see this woman because she had suddenly brought up about two ounces of bright blood. He diagnosed, no doubt quite correctly, Pulmonary Tuberculosis, and he treated her accordingly. It was then brought to my notice that previously to her consulting me she had been nursing an uncle who was supposed to have died after a pneumonia of seven weeks duration. This fact might probably have made me more careful of my diagnosis had I known it at the time. I was to blame however for not bringing it out through my omission in not taking a full history. Needless to say all cases of anaemia are not the same to me now; whilst even if all the information to be gathered, along with examination of the chest does not warrant one in immediately coming to a

diagnosis of Incipient Pulmonary Tuberculosis yet, if that patient does not quickly respond to iron and the general treatment of simple anaemia, the suspicion of tubercle somewhere comes on very strongly, to be settled as soon as possible may be with the help of the reactions of tuberculin and other methods to be discussed later.

COUGH & EXPECTORATION.

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Undoubtedly one of the earliest symptoms of active tubercle is ~~high~~ cough. It may be absent as in the case quoted above, and as in another also, to be mentioned under "Haemoptysis." Cough may be classed as one of the least inconstant of inconstant definite symptoms. It varies very much in different individuals, and in the same individual from time to time. It may be met with as the slight irritating cough without expectoration, as usually seen in early cases; or as the barking paroxysms accompanied with vomiting, sometimes seen in the lung tubercle of children, especially when associated with enlarged mediastinal glands. The physiological explanation is set down as being due to irritation of the vagus nerve endings, by the catarrhal secretion in the bronchial mucosa. The special character of the cough in children as mentioned above, and which is so often like whooping

cough is probably due to the enlarged glands pressing upon the trachea.³

Expectoration cannot be looked upon as one of the earliest symptoms and bears no definite relation to the cough; in fact the two often vary immensely with one another. In a case of phthisis expectoration to any appreciable extent indicates breaking down of the lung tissue, due to the action of the tubercle bacillus. When considering cough and expectoration in the case of children, one occasionally meets with that rarer form of Pulmonary Tuberculosis, where it forms part of a general miliary condition throughout the body. The diagnosis then, may not be easy because the symptoms and signs are generally like those of a bad Bronchitis. The child is exceedingly ill, dusky and cyanosed with rapid respiration; some cases have many of the appearances of Typhoid; the spleen may be enlarged as evidenced by percussion and the fever may be of the remittent type. The physical signs are those of râles and rhonchi all over the chest; no dullness or bronchial breathing to be detected. The prognosis is exceedingly grave, death usually resulting in about six or seven weeks. An example of this is seen in the following case:

N.W. age 4 years admitted to Monkenarmouth Hospital on November 12th. 1912.
HISTORY; Attacked with measles four weeks ago.

Never made a good recovery, having had persistent cough ever since. Two days ago became worse with rapid breathing and increased cough. Mother was advised to bring the child to hospital. The child had never been a strong one, was brought up on the bottle and was subject to attacks of convulsions from the age of six months up to about eighteen months. The mother was told the child had consumption of the bowels when it was two years old.

CONDITION ON ADMISSION. Child obviously ill; face dusky; temperature 104. Rapid breathing; twitching of the muscles of the face. Abdomen distended and tumid, veins well marked. On examination, coarse crepitant râles and rhonchi heard all over the chest. No definite dullness to be made out; no tubular breathing to be heard on auscultation, liver and spleen not enlarged. On ophthalmoscopic^{pic} examination, choroiditis evident in both eyes. Widal reaction negative.

The patient gradually became comatose and unconscious and died three days after admission. DIAGNOSIS. Tubercular Broncho-Pneumonia and Basic Meningitis.

POST-MORTEM EXAMINATION. Revealed the lungs studded all over with caseating miliary tubercles, one large one about the size of a walnut in the centre of the middle lobe of the right lung. Glands at the root of the lungs much enlarged.

and caseous. Spleen and liver slightly enlarged and also studded with tubercles. About one and half pints of yellowish fluid in the peritoneal cavity. Numerous tubercles present on the peritoneum. Examination of the brain showed injection of the pia-arachnoid and the presence of a yellowish exudate at the base of the brain extending forward as far as the optic commissure and backwards over the cerebellum and medulla; a few tubercles to be seen also in these regions but not to a marked extent.

Other cases are often met with not so typical as this. There may be a long standing bronchitis with fever from time to time, along with a history of a recent attack of whooping cough or measles. Râles and rhonchi are heard all over the chest. These facts should raise suspicions, but the child getting better leaves some doubt of the diagnosis. The following case is illustrative of this class of cases;

V.B. age 9 years. Admitted to Monkswearmouth Hospital on July 22nd. 1912.

HISTORY. An attack of whooping cough five weeks ago. Lately the spasmodic attacks of cough had given place to a cough of more constant character. Emaciation well marked.

CONDITION ON ADMISSION. Child obviously ill. Temperature 101; a slight degree of dyspnoea; a little cyanosis over malar eminences. Small

patches of dullness over both basal regions; a patch of dullness about the size of a five-shilling piece just below and to the right of the lower angle of the right scapula. Nothing abnormal to be detected at the apices. On auscultation there was well marked friction sounds over the special patch as mentioned; tubular breathing to be heard in parts, over both bases. Von Pirquet reaction negative.

DIAGNOSIS. Tubercular Broncho- Pneumonia and Pleurisy.

PROGRESS. The child did very well indeed under ordinary treatment and was discharged as quite recovered on September 4th. 1912. On her returning to report herself at the Out-patient Department about three months later, a Von Pirquet re-action was tried upon her; a very marked positive was the result.

The further consideration of Pulmonary Tuberculosis in children will be dealt with later on in a special chapter of the thesis bearing upon this branch of the subject.

Other persons about whom strong suspicions should be entertained are those who complain of 'Persistent Colds' or repeated attacks of 'Bronchitis', in which of course the cough is the symptom which they mostly complain of. They are usually thin weedy individuals who will not fatten, but apart from these so called 'Colds' are usually in quite fair health during the

intervals. Careful examination of many of these patients forces one to the conclusion that whilst not all, many of them are undoubtedly tubercular. The chest is usually of the long flat type with little expansion on inspiration and if no area of dullness can be detected, yet percussion often reveals one part or parts of less resonance as compared with the analogous areas on the other side. The nasal passages are often obstructed to a more or less degree. Enquiry often reveals a history of tonsils and adenoids in childhood which for some reason or other have not been removed. Further evidence as to the nature of these cases is provided when one enquires into the history of undoubted cases of Phthisis Pulmonalis; one often gets a history of these 'Colds' occurring before the alleged onset of the disease. The following case is interesting in this respect:

A.N. age 19 years. Clerk. Complained of palpitation, loss of appetite and general weakness. Was subject to attacks of bronchial catarrh with running nose usually about two or three times per year. As long as he can remember he has been subject to these. No cough in the intervals. Had a similar attack about three weeks ago which was rather worse than usual and does not seem to have picked up very well since.

EXAMINATION. Height 5'-8''. Weight 9st. 1 lbs.

Chest $30\frac{1}{2}$ " to 32", long and flat. Pale and anaemic looking, breathes with his mouth open. Tonsils enlarged. No definite dullness to be detected, but at right apex behind the resonance is less than that of the opposite side. Slight weakening of the breath sounds at both apices. ~~otherwise~~ otherwise nothing definite to be heard on auscultation.

The diagnosis was one of probably 'Incipient Active Pulmonary Tubercle'. I did not feel justified in notifying the case, so sent him over to consult the M.O.H. of the borough, who until the definite appointment of a Tuberculosis Officer was acting as such. He ~~diagnosed~~ disagreed with my diagnosis on the grounds that there was no cough or expectoration, nor anything abnormal to be heard on auscultation. However my advice to the patient was to stop work for at least three months and to get into the country if possible. He was given instructions as to treatment and general hygiene with special advice to carry out breathing exercises thrice daily in front of an open window. He faithfully carried out instructions and on coming back to see me four months afterwards had gained eleven pounds in weight he remarked that he had never felt better in his life. His chest developed from 33" to $35\frac{1}{2}$ ". I believe that he is now a recruit in Kitchener's Army. No doubt such cases are fairly common if looked for, and if attended to at once do very well. What is most important however is that the comparatively harmless latent tubercle is prevented

from bursting its bonds and becoming active.

HAEMOP^TYSIS.

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Hitherto signs of latent rather than active tubercle have been discussed and it might appear that one is premature in turning next to the consideration of haemop~~h~~ysis. Its argument for an early place in the discussion however is incontrovertible because it may be, and fairly often is, the first definite evidence to indicate the real nature of the disease which we are dealing with. (V.Cases 2 & 3) Seeing that the condition may be due to many other causes, it is imperative that its differential diagnosis also be considered. That it may be the first sign to open the eyes of the medical attendant is well seen in the case, I have already quoted above. I have admitted deserving of a certain amount of censure on that occasion but fortunately ample consolation is afforded me in the fact that many others have erred quite as gravely.

An initial haemop~~h~~ysis is often the means of causing a patient to consult his doctor. Over and over again one hears of the terribly alarmed person who hastily demands attention ~~attention~~ because a certain amount of blood has been expectorated. Fortunately when dealing with only small amounts, as is often the case, and with persons who never before suspected themselves of being consumptive, one can often

assure the patient that consumption is not the cause. It cannot be denied however that a large haemoptysis due to tubercle can come as an entire surprise to a patient who has hitherto always enjoyed apparently the best of health. The following case is a most interesting one in this respect:

R.W. Age 29 years. Police Constable of Ryhope near Sunderland. Admitted to the S.R.D. Isolation Hospital on August 25th. 1913. The history was , that one afternoon when walking through the village on duty, he suddenly coughed up quite a pint of blood; feeling faint he went into the nearest house. His medical attendant was sent for who came at once and testified to the amount of blood expectorated. The patient was taken home and put to bed where he quickly came to himself again. Later he was advised to consult Dr. McCrae , the county Tuberculosis Officer, who diagnosed the case as one of Phthisis Pulmonalis and sent him into the above Institution, where I saw and attended him. A further record of his case will be given later under the Appendix of this thesis. Striking feature about this case was that the patient never knew there was anything the matter with him until the haemoptysis occurred; he had always felt perfectly well previously.

When due to tuberculosis all grades of haemoptysis may be met with. It may occur at

any stage during the active course of the disease. As previously stated it may be the first sign to indicate the nature of the disease, or it may be the last and usher in a fatal termination after an illness of varying duration. (V. Cases. 26, 27, & 29)

Except in the cases of small haemorrhages the condition is probably indicative of excavation in some part of the lung; small haemorrhages may be the immediate precursors of a large one however.

It is nothing unusual for a case of phthisis to run its course from beginning to end without there ever having been any signs of haemoptysis. Probably the metaphor already quoted is very apt also on this occasion, namely it depends on the virulence of the seed and the fertility of the soil; certainly in chronic fibroid cases a marked degree of bleeding is very unusual.

Apart from small bleedings, rupture of an aneurysm in a cavity is the actual cause of the condition. There may be no apparent exciting cause or it may come on after some effort like coughing, straining, laughing or crying.

Patients who have had experience of previous attacks may be aware of premonitory signs e.g. a salty taste in the mouth or pain in some part of the chest.

The size and course of a haemorrhage

depends on many variable conditions such as the size of the aperture in the vessel wall, the size of the vessel, the proximity of the cavity to a bronchus, the blood pressure, the viscosity of the blood, on the treatment adopted, and on what is probably a most important point, the conduct of the patient himself.

The prognosis can only be gauged after taking into consideration the quantity and frequency of the haemorrhages, along with the degree of activity of the disease. Small haemorrhages are usually soon recovered from, a large one is a most serious symptom not only from the immediate loss of blood but because of the danger of the aspiration of virulent products further down the lung.

To estimate the degree of activity of the disease the behaviour of the temperature is most important. If this does not rise, or if owing to absorption of the blood elements it only rises to quickly fall again, then the outlook is favourable. If on the hand the temperature rises and keeps risen, this is an unfavourable sign indicating advancing disease.

In children according to Still,⁽¹⁴⁾ haemophysis is a rare condition and when it does occur is usually due to whooping cough or heart disease. Very often the haemophysis is of the spurious kind.

Under differential diagnosis the following

other causes of recurrent haemoptysis have to be considered:-

1. Valvular Cardiac Disease.
2. Aortic Aneurysm.
3. Certain Blood Diseases.
4. Vicarious Menstruation.
5. Various Non-tuberculous lesions in the respiratory passages-' Spurious Haemoptysis'.
6. Unknown Causes.

VALVULAR CARDIAC DISEASE.

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Rokitansky held, and probably correctly so, that all conditions which produce a state of venosity of the blood tend to impart an immunity to tuberculosis. (5)

Bier made use of this principle in the treatment of tubercular joint conditions. That the treatment is a good one and founded on true scientific principles no modern surgeon I think will deny.

Congenital heart disease seems to be an exception to the rule, and the reason for this is probably that we are dealing here with a protoplasm which has never fully developed its powers of natural resistance, so that a state of venosity is of very little help.

Most authorities rightly or wrongly credit Mitral Stenosis as being, next to phthisis pulmonalis, the commonest cause of haemoptysis

Dr. Price however makes the statement, "The most common cause of haemoptysis in pulmonary phthisis is Mitral Stenosis".⁽⁶⁾

Kidd states that although most writers agree that phthisis pulmonalis and mitral stenosis are never associated with one another, yet he has seen at least a dozen clinical examples.⁽⁷⁾ Samsom has stated that in all the post-mortem examinations made by him only two cases of phthisis pulmonalis associated with mitral stenosis came under his notice.⁽⁸⁾

Potain claimed that the co-existence of the two conditions was frequent.⁽⁹⁾ Dr. Byron Bramwell used to teach that a fairly common mistake was the interpreting of a haemoptysis as due to pulmonary tubercle when mitral stenosis was the cause.

Bandélier and Roepké sum up the position by saying "The teaching of Rokitansky that heart failure protects from pulmonary tuberculosis is not correct in this absolute form. It can only be said that mitral stenosis is accompanied by a diminished predisposition to tuberculosis, as is also and mitral insufficiency, when in consequence of congestion it produces brown induration of the lung. The progress of an already existing tuberculosis will not be checked by heart failure."⁽¹⁰⁾

In view of this conflicting opinion therefore it seems that more light is required upon the matter before a definite statement can be made. Personally I believe that phthisis pulmonalis and

mitral stenosis are rarely associated with one another; post-mortem evidence in this respect is better than clinical signs, and were the two frequently associated as the cause of haemoptysis, pathologists would not fail to point this out in their teaching; in none of the text books of pathology that I have perused is there any evidence that the two conditions are associated with one another. The reason for mistakes occurring clinically may be, that in the case of a patient coming with a history of cough and haemoptysis, crepitations may be heard at the apex of one or both lungs and so a diagnosis of phthisis pulmonalis be made. Cattle admits that he himself has made this mistake when after ~~ex~~ events proved him to be wrong. ⁽¹¹⁾ The following observation having some slight bearing upon this point is not without interest. Having, since the outbreak of the present war been appointed an additional examiner for H.M. recruits in this district I have noticed in the course of a few hundred examinations that quite a number of the candidates reveal the presence of a few fine crepitations when auscultating over the supra-clavicular areas. On percussion there is no dullness^{and} the ~~holes~~ are quite resonant. As most of the candidates are men of very fine physique from the neighbouring coal-fields and ship-yards, active tubercle as the possible cause of this condition in so many cases is out of the question.

What the cause of the condition is I am not at present prepared to venture an opinion, I merely mention the fact as being of interest as far as crepitations due to a non-tubercular cause are concerned.

When an haemoptysis is due to mitral stenosis the following facts to a greater or less degree are usually got. A history of acute rheumatism or chorea. On auscultation a slapping first sound with a marked presystolic bruit is usually heard in the mitral area. Along with this there is usually an accentuated second with marked reduplication to be heard in the pulmonary area. Persussion reveals increased dullness to the right with little or more to the left.

The haemoptysis in these cases is often alarming to the patient, and as said before is one of the causes for the patient hurriedly ~~as~~ seeking medical advice. When compensated mitral stenosis has been diagnosed as the cause of the condition, the patient may be consoled by informing him, that in his case the symptom is rather favourable than otherwise. This is because with good compensation the right ventricle is performing its work so well that the pulmonary blood pressure rises resulting in rupture of some of the small vessels into the air passages. The condition is far otherwise however when compensation has broken down;

blood in the sputum is then of fairly frequent occurrence associated with marked passive congestion in the lungs. The symptom is then of grave prognosis indicative either of thrombotic infarct of the lung brought about by atheromatous degeneration of the arterioles under high local blood pressure, or less frequently it may be due to embolic infarct, the result of an ante-mortem thrombus forming in the right auricle or right auricular appendix. (12)

AORTIC ANEURYSM.

 Copious haemoptysis has only two causes namely: Phthisis Pulmonalis and rupture of an Aortic Aneurysm into the air passages. The latter usually quickly ends in death, sometimes however there may be slight ~~haemoptysis~~ haemorrhages for a few weeks before the fatal termination, during which period the condition is usually diagnosed, as it is very seldom that a person suffering from aortic aneurysm is unaware of there being something wrong with him. Dr. Byron Bramwell however has recorded an example however in the case of a workman on the Forth Bridge when it was being built; he died suddenly without ever having suffered from any subjective symptoms. (13)

In the case of small bleedings when the cause of such is not definitely evident great care should be taken in the examination of the chest by the ordinary physical methods also by

X- Rays. The sputum if obtainable should also be diligently examined for bacilli. Tuberculin reactions should be tried especially if no evidence is obtained by other methods.

The following case of which a photo is appended was an intensely interesting one from a clinical standpoint. He came under my notice when stationed at Cleethorpes in 1907; my colleagues used to call him "A living text book of clinical medicine".

C.W. Age 47 years. Steam-trawler deck-hand. At the age of 19 years he contracted syphilis for which he underwent treatment for six weeks. He admitted to having been a heavy drinker especially between voyages; despite this he suffered from very little else until he reached the age of 39 years, when he began to be troubled with his legs especially at night time; he seemed to be losing control of them. His sight next began to fail him and he was then unable to work. He consulted a doctor and was told he was suffering from Loco-motor Ataxia; he improved for a time but was never able to resume his old work. He gradually fell back and now began to be troubled with a cough which was much worse when he laid down at night. He next noticed a heaving in his chest and that he was bringing up a good deal of expectoration often tinged with blood. He was close upon 46 years of age when he noticed that a lump was beginning

to form under his left collar- bone; at first there was a good deal of pain in connection with the swelling but lately this had not been so bad. He was now completely bed-fast and totally blind. Breathing was difficult and most relief was got when sitting up in bed and leaning forward. He has had no trouble with the bladder or bowels except with regard to the latter, constipation at times. There is no history of tightening pains or girdle sensation.

On examination the patient presented a very emaciated and worn out appearance; he was very happy and exceedingly intelligent and loved to talk about his sea-faring days with anyone who had a few minutes to spare with him. The large swelling was obviously an aneurysm involving the aorta and probably also the left subclavian; at time of examination it was only covered by the skin and fasciae, the bony structures have evidently been eroded through. He coughs a great deal and brings up a large amount of thick nummulated sputum well coloured with blood. Numerous T.B. present in the sputum when examined. Ophthalmoscopic examination reveals marked atrophy of both discs. The knee-jerks are absent. Sensations are all present. It is obviously impossible to test him for inco-ordination of the lower limbs, but with the finger to nose test he is fairly wide of the mark. His estimation of weights in the hands is

affected but not markedly so, e.g. he knows the difference between a fairly large book and a cigar box but he is out of it in the difference between a purse filled with silver and a small box filled with shells.

The diagnosis was one of Syphilis complicated with Phthisis Pulmonalis. Aneurysm and Loco-motor Ataxia.

The haemoptysis in this case was probably due to the tubercle bacillus because it was present more or less for the whole six months during which I attended him.

HAEMOPTYSIS due to BLOOD DISEASES etc:

- | | |
|----------------------------------|----------------------------------|
| 1. Pura. | 5. Pernicious Anaemia |
| 2. Scurvy. | 6. Hodgkin's Disease. |
| 3. Spleno-Medullar
Leukaemia. | 7. Malignant Specific
Fevers. |
| 4. Lymphatic Leukaemia. | 8. Haemophilia. |

These conditions deserve mention only, because they are all self-evident from the history, and clinical picture and examination of the blood.

VICARIOUS MENSTRUATION.

A posterior epistaxis due to this may occur with passage of blood into the air passages to be immediately coughed up again. The condition is rare. I personally never remember having seen a case in which I was satisfied that the haemoptysis was really due to this cause.

Careful attention to the history and physical signs ought to reveal the nature of the condit-

ion, but certainly this diagnosis should not be given until the patient has been under observation some time and possibility of tubercle and mitral stenosis absolutely put aside.

NON-TUBERCULOUS LESIONS.

When the more common causes of haemoptysis have been rejected as impossible then search must be directed for other sources of the haemorrhage.

According to Garel, the commonest of these other sources are varices at the base of the tongue. Garel collected 128 cases of Non-tuberculous recurrent haemoptysis and found this condition in 68 cases. An analysis of all the causes in these cases is not without interest, and is as follows:

Varix at base of the Tongue.	68.
Pharyngeal Haemorrhage.	3.
Albuminuria.	10.
Diabetes.	7.
Hepatic Conditions.	2.
Hysterical or Nervous Conditions.	12.
Arthritic Causes	5.
Menstrual Congestion.	3.
Unknown Causes.	18.
Total.	<u>128.</u>

A Varix is diagnosed by a pharyngoscopic mirror. A chronic pharyngitis may be the cause of a little blood in the expectoration and I have seen this occur in the case of a heavy smoker

who was also a gouty subject; in the mornings he was subject to heavy bouts of coughing and had great difficulty in clearing his throat, at the end of these efforts a little blood often appeared in the expectoration.

Haemorrhagic Laryngitis in association ^{with} Malignant disease, with chronic Brights' or Hepatic Cirrhosis may be a ~~cause~~ cause, laryngoscopic examination along with detection of the underlying condition will reveal the nature of the lesion. Similar conditions occurring in the trachea or bronchi are also a possibility of causing haemoptysis.

Frequent spitting of blood without much cough suggests the upper air passages as the possible seat of the lesion.

Too much reliance cannot be placed on the quantity of the blood as a fairly copious haemoptysis may occur from some of the other causes.

Other causes coming under this class are Gangrene of the Lung. Lobar Pneumonia. Injury to the Chest, and Malignant disease of the Lungs.

The extreme stench of the breath and sputum and the presence in the latter of a large quantity of elastic fibres suffices to diagnose Gangrene of the Lung especially if an underlying condition like diabetes is also known to be present. Lobar Pneumonia is evident from the history, physical signs and the viscid

tenacious character of the sputum. Injury to the chest by the history, only to be careful here that the trauma is the sole cause of the condition, and that it has not merely acted as a factor in producing bleeding from a tuberculous focus or an aneurysm.

Malignant Disease may be carcinoma or sarcoma, both usually secondary, very rarely primary. Diagnosis at first may be very easy or it may be very difficult. The primary seat of the lesion, e.g: Breast Oesophagus. Mediastrial Glands and Stomach etc may be known or easily detected.. A large effusion is usually present in the pleura, which is usually very markedly blood stained as shown by exploratory puncture; the sputum is copious and almost black in colour "Prune Juice". Microscopic examination may reveal particles of growth in the sputum or in the pleuritic effusion.

Other very rare causes of haemoptysis are those like Hydatid Cysts. Hepatic Abscess or Empyema bursting through the lung.

Sporotrichoses like Actinomyces. Distoma-Pulmonale. Gunia in the trachea or bronchus. All these are mostly characteristic in themselves and require only mention.

UNKNOWN CAUSES

Finally it must be confessed that a certain number of cases of haemoptysis must be classed under the "Great Unknown". In Garel's list the

number is as high as 18 in 128 cases or 14%. Naturally the greater the facilities there are at the disposal of the physician, the less the number of these cases will be. The number of cases correctly diagnosed in private practice must ~~be~~ of necessity be less than when the cases are admitted to a first class institution, where better observation and other facilities for diagnosis to be afterwards discussed, are available. Malingering must not be forgotten in connection with some of these cases, as I have known of a man ~~lying~~ trying to obtain sickness benefit under the National Insurance Act upon the plea that he was spitting up blood; the haemorrhage in this case ~~resulting~~ from sucking at a carious tooth.

Sometimes the pulmonary source of the haemorrhage in doubtful cases may be ascertained by skaking up some of the blood clots with a little water in a test-tube; decantation and renewal of the water to go until the clot is decolourised. The Little white pellets which may be left are then examined microscopically and if of pulmonary origin the strands of fibrin seen, ^{may} correspond to the small bronchioles. (Mann.)⁽¹⁵⁾

PAIN.

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Is not a constant feature in phthisis, although very few cases pass to a fatal termination in which pain has not been present at one time or another. There is no relation

between the extent of the lesions present and the degree of pain. The lung itself is not the source of the symptom, as very large excavations can occur without the patient ever complaining of pain.

Pain is usually due to pleurisy, to a pneumothorax, to an accompanying bronchitis, to an intercostal neuralgia, or indirectly to the violence of the coughing dragging upon the diaphragm and chest muscles.

In the case of Acute Bronchitis it is felt more as a kind of rawness or a ~~severe~~ sense of oppression extending from the manubrium sterni downwards; lateral pain may also be present due to the strain of coughing.

When a pneumothorax is the cause, it comes on suddenly and is of a most excruciating character referred to the whole side of the thorax, it is accompanied by a great deal of dyspnoea owing to the sudden collapse of the affected lung before its fellow on the opposite side has adjusted itself to the new conditions within the thorax. It is possible for a pneumothorax to occur without pain, the sense of oppression, the collapse and the dyspnoea are present however.

Intercostal neuralgia frequently occurs, as in other debilitated subjects. Respiratory movements may be reflexly restrained for the time being. The absence of fever and friction

sounds suffice to establish a diagnosis.

When pleurisy is the cause of the pain, it is always referred to the affected side. Deep respiration and coughing always aggravate the condition, whilst immobilisation of the ribs usually diminishes it. The pain is usually of a sharp, stabbing, lancinating character, and on auscultation friction sounds are usually audible, although I have seen a few cases of undoubted pleurisy where the friction could not be heard; the very fineness of the sound may possibly allow of its escaping detection.

That pleurisy may occur without pain is also possible, because I have also often detected friction sounds when examining a patient who has made no complaint of pain. The pain again may be referred to other parts of the chest away from the pleurisy eg: in some cases friction sounds may be heard in the axilla, while the patient complains of pain pleuritic in character over the lower ribs.

The onset of effusion often moderates the pain very much indeed, or it may cause it to disappear altogether; this is probably because respiration no longer results in the inflamed surfaces of the two layers of the pleura rubbing against one another.

A history of pleurisy is often obtained as the alleged starting point of the onset of

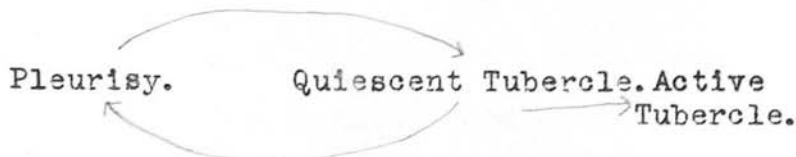
pulmonary tuberculosis; some authorities have gone so far as to say that all pleurisy is of tubercular origin. Whilst not quite prepared to go so far as this there is no doubt that many cases of pleurisy are undoubtedly tubercular; the mere fact that the patient recovers quickly is not to my mind an argument to bring against it being of a tubercular nature.

Tuberculosis of the pleura is probably always secondary to infection in some other part of the body. It may be ~~in~~ the neighbouring structures like the lungs or mediastinal glands which are the primary seat of the lesion, or this may be in the abdomen eg: the mesentric glands or the kidneys. (V. Case 14.) The primary seat of the lesion may not be found clinically, yet this does not contradict the secondary nature of the pleurisy. Co-incident with ^{the} assumption of this fact, that class of pleurisy which was ~~fo~~ formerly known as "Idespathic" has largely diminished in size or vanished altogether. An appearance of apparently robust health is no criterion to judge ~~a~~ ^{the} non-tuberculous nature of a case. The more frequent method of pleural invasion however is by direct pulmonary extension, usually from some focus superficially situated in the lung, and so in close proximity to the serous covering; it is imppssible not to conclude in such cases that the pleurisy is tuberculous.

In other cases where the lung conditions are not evident at first, the course of the disease must be carefully watched before the nature of the disease can be definitely settled as tubercular. In such cases the cough is prolonged well into convalescence. Marked debility along with night sweats in an attack of pleurisy is very suspicious; haemoptysis also may make its appearance. When convalescence sets in, it is slow and protracted and now for the first time definite signs may show themselves in the lungs; it may be in the lung of the opposite side to the seat of the pleurisy.

The onset of an attack of Acute Military Tuberculosis may commence in an attack of pleurisy; the signs and symptoms then go rapidly from bad to worse.

Pleurisy often occurs in persons suffering from quiescent, or may be latent tubercle of the lungs. The nature of a vicious circle may then be established i.e. the tubercle accounts for the pleurisy which then reacts on the quiescent condition on the lung lightening it up:



Effusion into the pleural cavity may be present, then the tubercular nature of the disease may be gathered from examination of the other lung; or a primary focus may be discovered elsewhere

eg: Kidney or Joint. Physical signs at the apex of the compressed lung are not reliable as these are often found to disappear on reexpansion of the lung after aspiration. Examination of the exudate direct for T.B. is usually negative, the absence in the film of other micro-organisms indirectly infers the condition to be due to the tubercle-bacillus. An inoculation experiment usually settles the diagnosis.

On going over my notes I can recall 16 cases of Pleurisy with effusion made up as follows:—

Complicating Acute Pneumonia	3.
Carcinoma of the Lung.	1.
Tuberculosis.	10.
Uncertain.	2.

The effusion was diagnosed as certainly present in all cases except two by exploratory puncture and the underlying causes by clinical signs only. With the exception of the case due to malignant disease I am prepared to admit that all the rest may have been associated with tuberculosis latent or otherwise. In the case due to carcinoma (V. Case 4.) the primary seat of the lesion was probably in the posterior mediastinum; there was regurgitation of food on some occasions, but this symptom was not marked enough to warrant a definite diagnosis of malignant disease of the Oesophagus.

Of the three cases following pneumonia,

two eventually turned purulent, one of which died shortly after operation; the other two made a very fair recovery.

Of the ten tuberculous cases, four were in cases known beforehand to be consumptive; in the other six, there were signs of mischief in the opposite lung in two ; whilst of the remaining four, ~~one~~ was associated with an acid urine containing pus and blood, (tubercular ~~kid~~ kidney) another with a tubercular ankle ^{joint}, whilst a third was associated with tubercular mesenteric glands. The last was an interesting case of Acute Miliary Tuberculosis which was sent into the S.R.D. Isolation Hospital as a case of Typhoid , and who died 36 hours after admission. On postmortem examination I found a condition of acute tuberculosis throughout the body, the right pleural cavity being full of yellowish fluid. (V. Case 13.)

A synopsis of all these cases will be given in the Appendix.

ALTERATIONS in the VOICE.

 Apart from tuberculous infiltration of the larynx, alterations in the voice often occur in the course of phthisis . (V. Case 17) How often has one heard consumptives bewailing the fact that they were good singers before the disease attacked them. This is not because of loss of power only but because the quality of the voice has also altered.

A certain^{degree} of hoarseness is occasionally an early symptom but it may occur at any stage of the disease; it may remain entirely absent however. These symptoms ie: lack of power, tone and hoarseness, may be due to weakness of the adductor muscles of the larynx, exactly analogous to the weakness that occurs elsewhere throughout the body.

The most important point to remember is, that they may be due to a commencing tuberculosis of the larynx. Swelling and thickening of the cords are the first changes and which account for the hoarse character of the voice. Finally ulceration and destruction of the larynx may set in and so form one of the most distressing features of the disease.

It must not be forgotten that the possibility of a recurrent laryngeal nerve becoming implicated in a pleuritic thickening about an apex, may occur; this will result in one of the cords becoming paralysed. I have never seen an example of this.

DYSPNOEA.

Like all the others, breathlessness is a variable symptom. It may be the result of actual diminution of lung substance, or from irritation of the vagus nerve endings by the destructive processes. The latter probably accounts for the asthma-like attacks which sometimes occurs in phthisical patients.

(V. Case 18.)

The severity of the dyspnoea bears no relation to the amount of lung destruction, as great excavation may occur with little dyspnoea and vice versa. Probably the rapidity of progress of the disease plays a large part in the causation of this symptom, adaptation has not time to take place. If destruction is rapid then the greater^{is} the likelihood of dyspnoea setting in. When destruction is slow then breathlessness is not so likely to occur until close upon a fatal termination.

A weak heart due to an enfeebled condition of the cardiac muscle wall is often associated with^{dyspnoea} as evidenced by the palpitation and rapid pulse that occur when a consumptive makes slight exertion; a certain amount of cyanosis is also sometimes to be noted on these occasions.

A considerable degree of anaemia may also accompany the symptom; this is probably due to deficient oxygenation of the tissues also to a toxæmia. That anaemia alone will not account for the symptom, is evidenced by the fact that when the blood is examined, often a large degree of dyspnoea present, ~~also~~ does not correspond with the small changes as found in the blood.

The nervous element probably plays a part also, because the symptom is much more frequent in neurotic subjects, than in those of a more stolid nature.

FEVER.

Because of its great importance in estimating the degree of activity of the disease and also the prognosis, fever is one of the most important symptoms in the course of Pulmonary Tuberculosis. Extreme sensitiveness of temperature is characteristic of tubercle. Slight extra exertion may bring about a rise in temperature, and this also is enough to make one suspicious of the presence of the disease.

(V. Case 20.)

The manner in which the fever reacts to treatment is a most important point in estimating the prognosis. If the fever keeps up continuously in spite of all means adopted then the case can be looked upon as hopeless; on the other hand if treatment results in a fall of the temperature which is continuous, then a more satisfactory view of the case is justified,. There are exceptional cases to this however which are sometimes met with, chiefly amongst those of the chronic [p]broid type, which gradually retrogress towards a fatal end without ever at any time exhibiting much pyrexia. I have noticed much the same kind of thing happen in some cases which have been treated with Spenglers Serum. (V. Case 21.)

The causes of the fever, or rather the manner in which the causes act, are far from being completely understood. Undoubtedly the chief

cause is the toxins generated by the T.B. themselves; but added to this, other important agents are the products of tissue destruction and the disintegration of the white blood corpuscles; moreover, the result of entrance into the body of secondary organisms is a most important factor. Why should we get pyrexia in some cases and not in others, which otherwise are apparently similar in every other respect? How are the different forms of fever to be explained? The temperature may remain elevated for weeks, then drop to normal and keep so for weeks; Why is this? Why should it fall in the morning to rise in the evening in so many cases? How is the lack of relation between its presence and the various stages of the disease to be explained? As far as I know the greatest of our authorities have failed to explain all these characteristics.

The practical bearing of the symptom is its importance as an aid to estimate the prognosis, and also the effects of various therapeutic agents. It has been denied by some that the T.B. alone can cause fever. The evidence of the fever in cases of pure military Tuberculosis, and the effects of injections of large doses of tuberculin (a product of T.B.) into the afebrile subject, is I think enough to contradict this statement.

There is no doubt however that in many

cases secondary organisms play a more important part in the production of fever than the bacilli themselves; chief amongst these are the staphylococcus, the streptococcus, the influenza bacillus., the B. Pyocyaneus and the M. Tetragenus; of these the first two are the most common.

The degree and course of the fever depends largely on the sensitiveness of the heat Regulating Centres eg: upon the nervous system. Pyrexia is often classed as follows:

- A. Sub-Febrile or slight rises only appearing from time to time as in the evening.
- B. Intermittent. Higher than the former but returning to normal at some period of the day.
- C. Remittent. High fever, only dropping a point or two from time to time, and never then to normal.
- D. Continuous. High fever exhibiting little variation even from hour to hour.
- E. Hectic. Is a high evening temperature with a large morning fall, sometimes to below the normal. This may go on for months and is a very characteristic form of fever in advancing cases.

The above classification is more an ordinary arbitrary one for the sake of description than real. The "Inverse" form may be present ie: a morning rise and an evening fall; or again a very irregular chart may be noticed conforming to none of the above types in its entirety.

There is thus then no type of fever which is pathognomonic of tuberculosis; all that can be said is that a continued high fever is a

sign of advancing disease. The chart indicates any changes which may be taking place and which otherwise might escape detection.

NIGHT SWEATS.

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Another variable symptom, but very significant when it does occur. It is in some cases more the subject of complaint from the patient than any other symptom.

The patient may complain of the whole body being bathed in perspiration every night; so profuse sometimes that the clothes are soaked. In other cases only slight moisture of the hands, feet or scalp may show itself. Why the symptom should be so marked in one case and ~~ab~~ absent in another, apparently identical in every other respect, is inexplicable.

Just as in malaria the fall of temperature is accompanied by profuse perspiration so in phthisis the same thing often occurs. The effect of the varying temperatures on the controlling centres in the brain may be the cause of this, and so be nature's attempt at resisting the effects of prolonged fever. It is well known that evaporation and loss of moisture from the body surface is the great way of controlling the balance between heat, generated, and heat lost. As stated before the heat regulating centres are markedly sensitive, probably owing to toxin effects from the T.B.; now as the centre controlling perspiration is a part of

the heat regulating centres, one is justified in assuming that this centre also becomes markedly sensitive of the toxins generated, so that a pathological reaction results from the exaltation of a physiological one.

If the sweat centre then is in a state of pathological activity, or in other words in a state of extreme hyper-sensibility, small influences may produce a large effect. To use a metaphor again the normal person may be compared to a tradesman's scales and the consumptive to the delicate balance of the physicist, as far as the sweat centre is concerned.

Carbonic Acid accumulation in the blood may be an irritant of the sweat centre; this can be argued from analogy with the clammy sweats that occur in asphyxiation. Increased CO_2 accumulation naturally occurs during sleep when katabolism is at low tide; this coincides with the time at which the sweats make their appearance i.e. the increased CO_2 has stimulated the cerebral sweat centre. The close association between pyrexia and night sweats can be explained. It also explains how it is that a consumptive perspires freely on the slightest exertion i.e. increased CO_2 production occurs on exercise. There is no doubt also, that an increased amount of toxin



also passes into the circulation during exercise. This is the principle underlying the method of treatment by "Graduated Exercises"; the patient practically made to manufacture his own tuberculin.

CIRCULATORY SYSTEM AND SYMPTOMS.

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It has comparatively recently been demonstrated that tubercle bacilli are often present in the blood of consumptives; therefore symptoms something after the nature of a septicaemia may be looked for.

Actual blood changes are those of an anaemia, and due to this we must at least partly ascribe such symptoms as Pallor of the Skin, Anorexia and Palpitation.

Weakness of the heart's action occurs and is manifested by a fall in blood pressure, a feeble accelerated pulse and palpitation. This is probably due to toxic action on the heart muscle.

A fall in blood pressure whatever the exact cause, is probably a usual manifestation of active mischief in the lungs; this, in the examination of 80 cases of ambulatory phthisis ~~tuber~~ pulmonalis at the Borough Tuberculosis Dispensary in June 1914 not one of them exceeded 110 mm of Hg; some of them indeed fell as far as 60 mm; the average worked out at 89 mm of Hg. I do not know whether this condition can be ascribed as specific to pulmonary tuberculosis

or not. Before deciding this one would first wish to know the average blood pressure collected over a good many cases in many other diseases such as Chlorosis, Typhoid, Diabetes Mellitus and so on; indeed one would not be surprised to find that in these also, there was a fall in the blood pressure, although I have no definite data to go upon. It is for this reason then, that as yet, I do not look upon the phenomenon as of so much importance as Spengel seems to give to it; had he composed his findings in Phthisis Pulmonalis with the findings in other diseases and found a considerable degree of difference, I should have looked upon the matter as much a more important symptom of pulmonary tuberculosis than I regard it at present.

In the last stages of consumption, great dilatation of the right heart and the presence of ^{bruits} ~~bruits~~ at the mitral aortic and pulmonary areas may be detected; these defects are probably due to actual myocardial degeneration. A water-logged condition throughout the body often occurs shortly before death and indeed it is not uncommon for a phthisical patient to die from heart failure rather than from effects more directly attributable to destruction and loss of lung substance. (V. Case 21.)

In the case of the more chronic fibroid cases, changes in the right heart often occur

analogous to these which occur in chronic bronchitis and emphysema; cyanosis, albuminuria and many of the signs of backward pressure may make their appearance long before a fatal termination to the illness occurs.

GASTRO-INTESTINAL SYMPTOMS.

Loss of appetite has already been mentioned as a possible early symptom of the disease. It is not a constant symptom and indeed increased appetite may occur in some cases; this however I am inclined to think is rather a psychical result met with in those who have an idea that they may be developing consumption and who hope to ward it off in this way or as Terence puts it "*Eflamma pretere cibum*"

Loss of appetite is a most important symptom however, inasmuch that it may interfere with the necessary treatment in the early stage.

It is probably due to toxic causes, the toxins checking the due secretion of the "Appetite Juice" of Pawlow". In later stages, along with this anorexia there may occur pain after food, distension, nausea, and vomiting, i.e. the speedy pain and speedy vomiting of Byllie, due to toxic influences. (V. Case 22.) Such symptoms are often the cause of the patient first seeking medical advice, and if the chest is not carefully examined a wrong diagnosis of a serious nature may easily be made.

Sometimes owing to the severity of the cough, and quite apart from the irritability of the stomach, vomiting may take place, especially after a meal. Another "vicious circle" may again be established as follows:

Cough. Full Stomach. —→ Vomiting

Irritation of the vagus and its connections may possibly account for this syndrome.

With regard to the bowels, constipation often occurs, but it is not unusual for the bowels to act with perfect regularity even up to the last stages; towards the termination however, alternating constipation and diarrhoea often sets in.

SYMPTOMS Connected & The NERVOUS SYSTEM.

The onset of tuberculosis is often marked by changes in the psychological condition of the patient; he may exhibit irritability, weakness of will, or ^{di}moodiness (V. Case 23.) Optimism is often well marked even in those cases which are rapidly going from bad to worse; the "Spes Phthisica" is often present unto the last. The reason of this may be that the patient does not fully realise how close upon him, death really is.

Pessimism when met with is usually the result of a stoical ^{li}indifference to everything. The patient knows that he is not improving after having clutched at almost every straw in

the hope of finding a remedy; baffled he falls back in despair, usually a courageous despair however, taking the form of an apparent pessimism. One is often filled with wonder at the fortitude with which such patients knowingly ~~face~~ a death creeping upon them by inches.

A high intellectual standard is often to be met with in phthisical patients; indeed in some cases I have often imagined that the mental faculties are in a state of stimulation so much above the average does the intelligence appear to be.

Vasomotor changes often occur from the beginning; such are rapidly changing colour of the face at times, pallor giving place to a deep blush which shortly falls away again; a feeling of heat or cold in different parts of the body may occur with some patients.

Tubercostal neuralgia has already been mentioned under pain. Facial neuralgia may set in and be a very difficult complication to treat. Such symptoms ^{are} probably due to toxic influences acting on the nerves.

Unequal pupils are sometimes said to occur due to involvement of the Sympathetic about one apex. I have never seen a case of this personally; pupillary changes however should suggest them, and a thorough examination of the

chest in the absence of other more likely causes.

The Brain, Spinal Cord and Meninges may all be secondarily affected as so often seen when dealing with children.

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PHYSICAL SIGNS.

"Auxilium non leve vultus habet "

(Ovid.)

It has been well said that no two cases of pulmonary tuberculosis are alike. The great diversity of symptoms which may present themselves has already been considered. Understanding then, that these must have their foundation in some pathological condition within the thorax; which condition may also reveal itself in countless different forms when laid bare on the post-mortem table, one indeed need not be surprised at the multiplicity of the physical signs, ^{which} may be encountered in the clinical study of Phthisis Pulmonalis.

Although these signs may be classified into more or less well defined groups, when considered from one point of view only, e.g: Inspection; it must not be forgotten that this is one, and only one aspect. We have still palpation, percussion, and auscultation to reckon with, and these again under varying conditions of time, rest, exercise etc; whilst anatomical differences depending on age and sex need only be mentioned.

One can begin to realise then what little reliance is to be put upon any one sign when

taken away from its context. It is only after all the evidence has been carefully sifted and weighed, that one can truly say that a given case is, or is not, one of pulmonary tuberculosis. Neglect of the principle is a source of many errors resulting not in a diagnosis of 100% of the true cases submitted, but a rate of 120 or even 150%; meaning thereby that many are labelled "Tuberculous" who are not, and who should not be so named. What comparative value again is to be put on the various therapeutic measures, when at the onset the statistics may be so grossly vitiated? No wonder then that such a storm of controversy may arise, as is at the present time thundering around "Tuberculin" in treatment. Far be it from me however to suggest that errors in diagnosis are the sole cause of such a state of affairs; my intention is only to point out what may be a partial cause. Well I know that where one specialist whom we may call "Treatment" will accept almost every case sent to him as a case of tubercle, another whom we may call "Diagnosis" will reject a large number of cases, not on the grounds that they are cases of tubercle but because they are not "Active" tubercle, a very different thing indeed. However erudite and experienced a clinician may be, it is quite impossible with some cases for him to say offhand that a certain patient is suffering from

active tubercle, or even from tubercle at all; prolonged observation may be absolutely necessary to settle the diagnosis.

Therefore in view of all these facts, I should hold that in the end, it is on the general practitioner that a great deal of responsibility falls for the clearing up of many important points relating to the "Control and Eradication of Tuberculosis"; I hold also that the diagnosis of early active tubercle is essentially within the province of the general practitioner; if then this diagnosis is based upon insufficient evidence deduced from a few physical signs only, the chances are that many mistakes will occur. As an example of what I mean the following case is quoted.

A near relative of my own, age 25 years, whom I had not seen for two months complained of having had an attack of influenza about three weeks previously, and that since then she had been troubled with a cough. On examination of the chest what struck one at once was a hollowing over the inner end of the 3rd. right interspace about $2\frac{1}{2}$ " by $1\frac{1}{2}$ " in size; there was nothing else abnormal to be detected; a brother shortly before this had died of Phthisis. Taking into consideration the history, the cough and the fact that the patient was thin and very much run down at the time her medical attendant had become suspicious

and had advised me to get further advice. I myself agreed with him entirely. Dr. Gulland of Edinburgh was kind enough to see the patient for me. His opinion was that the case was not one commencing phthisis and that the hollowing was due to a congenital absence of a slip of the Pectoralis Major; the cough was probably due to a simple bronchial catarrh. He was, as after events showed, quite correct; the patient getting perfectly well after a few days holiday in the country. The hollowing in the chest had undoubtedly always been present, although previous to this it had passed unnoticed. Six years have elapsed since then, the patient is in perfect health and the hollowing is still present.

The faulty interpretation of various combinations of physical signs, along with bad technique in the mode of conducting an examination, are probably more than any other causes, responsible for many errors of omission as well as commission.

Although no single sign may be said to definitely characterise any one lesion in the thorax; yet the manner in which several associated signs group themselves together, along with several essential facts gathered from the history and symptoms, will allow of the differential diagnosis of most of the various possible conditions.

Physical signs are the objective evidence elicited by the examiner, as contrasted with the

subjective evidence described by the patient, and which has already been discussed.

Apart from the assistance provided by the use of the X-Rays and Laboratory, the usually the means of examining the chest clinically, comes under the following methods:-

1. Inspection.
2. Palpation, including
Mensuration.
3. Percussion.
4. Auscultation.

INSPECTION.

As an aid to diagnosis inspection is often of great value. It has already been mentioned how our clinical forefathers ~~were~~ were so adept at pointing out to us the features of the so called "Tubercular Diathesis". With all due weight to this, there is no doubt however, that when inspection alone is relied on, as it usually is in the so called "Lightning Diagnosis", serious mistakes often result. This might have been in the case just mentioned, and would ~~certainly~~ certainly have occurred in the case of the policeman already quoted before, had he been seen before the onset of his haemoptysis.

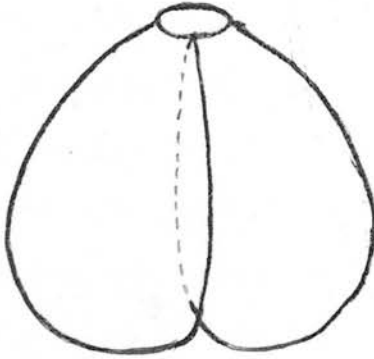
We may have "Chests" revealing many of the appearances, so often met with in phthisis, and yet when the evidence is complete the diagnosis absolutely negatives active tubercle. Or again

we may meet with a thorax almost Herculean in shape and size, and yet no other evidence than that the subject is a victim of the tubercle bacillus can be come to. It is evident then that "Inspection" must at once be assigned its true place, and that is as an aid, and an aid only, to diagnosis. Recognising this from the beginning then evidence of much value may be deduced from it.

Assuming that we have already had the history and symptoms before us, then evidence corroborative of many points in this may be obtained. For this it is absolutely necessary that the patient should be stripped to the waist; a sitting or a reclining posture is optional, care being taken however that if the latter is adopted that the body rests equally upon the hips and shoulders. In the case of a female it is advisable that a light wrap be placed over the shoulders when the front of the chest is examined, which again can be put over the front when the back is exposed. To this end I have personally designed a very simple and useful wrap for the examination of female patients in the consulting room. It consists of a flannel cape and rather full. There are no button attachments to it, it being simply fixed to the patient's neck by a circular band with attached hooks. The opening can be brought to the front or back quite easily without its

Conclusion

having to be taken from off the patient. The chest is well exposed for examination either back or front by simple throwing the edges of the cape over the shoulders.



Such signs as the amount of emaciation and general appearance of the patient are at once apparent. The appearance of the face is noted, whether pale, sallow, flushed or dusky. The condition of the lips and conjunctiva, as an indication of any degree of anaemia; the presence of cyanosis, clubbing of the fingers; are all rapidly noted in one's mind.

Cyanosis may be due to lung or heart trouble; when met with in phthisis it usually indicates changes in the circulating system. Oedema, or puffiness below the eyes may be apparent and will at once suggest in addition, an inspection of the regions around the ankle joints and also the abdomen.

The character of the breathing will be carefully noted, whether dyspnoea is present or not and if so whether inspiratory or expiratory in type. The type of respiration is noted:

Thoracic, Abdominal, Abdomino-Thoracic, Thoracies, Ab
 Abdominal, bearing in mind the influence of sex here.
 Movements of the alae nasal and larynx may here
 afford valuable evidence. Are the accessory
 muscles of respiration being used? What is the
 condition of the skin, Pigmentation. Eruption.
 Pityriasis Versicolor?

As regards the general form of the chest,
 it must be remembered that like individuals, no
 two are exactly alike; yet striking variations
 may be at once apparent. The effects of occupation
 nasal obstruction and rickets may be seen and
 noted.

Although certain shapes of chest are
 commonly regarded as associated with intrathoracic
 disease yet the evidence here must be taken for
 what it is worth, along with the consideration
 of the rest of the evidence.

A characteristic ~~namely~~ of chest is known
 as the "Phthisical" or "Paralytic" because it is
 frequently associated with pulmonary tuberculosis.
 Here there is a flattening in the antero-posterior
 diameter with widening real or relative in a
 lateral direction; the sternum is depressed whilst
 the clavicles are prominent especially towards
 their sternal ends; the neck looks long. The ribs
 are prominent and the intercostal spaces sunken
 and narrower. Behind, the scapulae stand out in
 bold relief from the ribs, giving a so called

"Winged" appearance- "Pterygoid" or Alar" chest. On deep breathing the front of the thorax lifts bodily as a whole, not rising and swelling equally all over, in the graceful manner of the healthy chest.

Although this type of chest is frequently associated with phthisis, it must not be forgotten that it may be met with, where there is no intra-thoracic disease, and also advanced phthisis may be met with, with none of these appearances. Indeed pulmonary tuberculosis is far commoner in chests that show none of these changes.

Local changes must be noted in an otherwise normal chest eg: Local flattening or bulging; the presence of such areas if accompanied at the same time with deficient respiratory movement over them, is exceedingly suspicious of existing underlying disease.

"Delayed or Lagging" local movements may be observed. This may occur over parts which are quite as full in appearance as the rest. This form of movement on respiration, unlike retraction with deficient movement occurs in quite an early stage of active disease and is therefore of great diagnostic significance. As the disease advances the lagging movement disappears, and only deficient movement remains. Bilateral disease causes great differences in movement on the two-sides. Over the site of the old disease there is

deficient movement, whilst over the more recently affected side there may be full but delayed movement.

The amount of local retraction present practically indicates the amount of lung contraction due to fibrosis which has occurred. The age of the patient plays an important part here however. A chronic infection settling in, in a young patient is of necessity bound to be accompanied by a greater amount of thoracic shrinking than in the case of an old subject where the thorax has lost much of its elasticity. The greatest amount of retraction occurs in younger subjects and where the lesion is large and mostly unilateral; then the accompanying signs may be displacement of the heart, as indicated by the position of the apex beat; and may be also, a certain amount of lateral spinal curvature. Hence these points should be specially looked for and if found should raise strong suspicions.

The whole, or almost the whole of one side of the chest, may be apparently smaller or larger than its fellow; this along with absence or marked deficiency of respiratory movement usually indicates in the first case a pleural effusion, in the latter a pneumothorax.

Undue rounding "Barrel" chest or angularity "Pigeon" chest, suggestive of ~~an~~ accompanying Emphysema, and Rickets respectively may be noted.

①
 LITTEN'S SIGN is supposed to have great diagnos-

 tic value; unfortunately it is only well establi-
 shed in a few cases. To elicit this the patient
 must be lying in bed in a good light and with
 his feet directed towards the best light.
 The examiner stands by the head of the patient
 who is told to take as deep and as slow a
 breath as possible, when a shadow descending
 from the 7th. to 9th. ribs may be observed,
 which recedes again on expiration. The
 explanation of the shadow is, that it is due to
 the descent of the diaphragm on ⁱⁿspiration.
 It is not capable of recognition unless the
 physical conditions present allow of full
 expansion of the lung eg: Extensive pleural
 adhesions, ascites, peritonitis, and such like,
 prevent its occurrence. The sign is of diagnos-
 tic import in early cases of phthisis eg: where
 one apex only is involved when the shadow
 observed on the affected side is much less than
 on the other. My own experience of first looking
 for this phenomenon was not a success and I was
 inclined to think it was of no value; further
 practice and experience however has forced me
 to the conclusion that it is of certain value
 in a few cases especially those who have not
 too thick ~~and~~ thoracic wall. It ought certain-
 ly be looked for in many of those cases where
 the physical signs are somewhat scanty.

Under "Radiography" the diaphragmatic movements will be again discussed; here often phenomena which are practically a magnified "Litten" are easily detected by the use of the fluorescent screen.

Evidence of tuberculosis in other parts of the body should be looked for. Much assistance is afforded towards a diagnosis when such conditions as Tuberculous Glands in the Neck are evident or there may be the old puckered scars that they were present in childhood. Tuberculous joints may be present. Tubercle may have been lying latent in some part of the body for years only to light up again on the first available opportunity.

In the case of Acute Miliary Tuberculosis, the bones, skin, testicle and joints are often *the* primary seat of the disease.

Enlargement of the thoracic veins, especially in children, is often an important sign bearing upon the presence of intrathoracic disease, especially "Enlarged Tubercular Glands". The explanation of this is probably that obstruction of the flow through the Azygos veins occurs due to pressure by the enlarged glands. The Azygos Major which also receives the Minor, arising in the abdomen runs upward through the posterior mediastinum as far as the root of the lung when, lying above this, ~~is~~

it runs forwards to terminate in the Superior Vena Cava. The function of these veins is to drain all the intercostal spaces except practically the upper two or three on the left side of the chest, where the intercostal veins run more or less directly into the left Innominate Vein.

② "Still" states that a specially large vein is often seen passing along, from just below the coracoid process to the inner side of the second interspace. This sign he says, is of chief value when seen on one side only. Indeed I claim that it can only be of value when this is seen on the right side only; on the left side this vein as stated above terminates in the left innominate which is not likely to be obstructed by enlarged glands, because unlike the Azygos Major it has no direct relationship to the root or apex of the lung and the posterior mediastinum. It is often stated that these veins on the right side are more often enlarged than those on the left simply because commencing phthisis pulmonalis is commoner on the right side; whether this is so corresponding to anything like this extent or not I am certainly not prepared to say; but this I know that when the veins in the upper part of the left side of the thorax are to be observed enlarged either in themselves alone or sharing

in a general enlargement of all the intercostal veins, other causes then commencing mischief about an apex or enlargement of the posterior mediastinal glands must be looked for. My reason for this is based on the anatomical grounds already stated above. I have never seen this explanation given so in any text-book but I give it for what it may be worth.

Other signs of lesser importance to be looked for are inequality of the pupils, a high palatal arch, lowering of the nipple on the affected side, and atrophy of the breasts with diminution of the areolar pigment. Any of these conditions may be associated with phthisis pulmonalis, but they are inconstant and often also associated with other conditions, so too much weight even when found should not be attached to them.

The condition of the teeth is more important however, not so much as a diagnostic sign, although a foul state of the gums is often associated with pulmonary tuberculosis; but because the chronic toxaemia resulting from such carious teeth or a pyorrhoea alveolaris is often not unlike the general condition and appearance associated with early active tubercle and which may be difficult to diagnose with certainty.

Even if this is not the case the tubercle-

bacillus is an organism which flourishes in the symbiotic condition with other pathogenic germs, and hence from a treatment point of view careful attention to the teeth will in all likelihood bring about a less virulent condition of the T.B., and at the same time elevate the resistance of the patient generally, e.g.:

"The soil becomes less fertile and the seed less active". From my own experience I have often thought that many of the patients sent to a tuberculosis dispensary for observation and treatment might not be tuberculous at all, and that the condition of the mouth would account for most of their symptoms. There is some reason to suppose then, that due dental treatment might result in the diminution of not a few doubtful cases. Very few of the type of patients met with in a dispensary can be said to have a clean mouth; indeed it is the exception to find such amongst the lower classes generally, although they may be quite unaware of the fact because their teeth give them no pain, and they can chew their food well.

Much the same arguments apply to the condition of the nasal passages which should be inspected in all cases. Tonsils and Adenoids, Necrosis of the Turbinates, Sinusitis should all be looked for.

(44)

③

TURBAN'S SIGN.

Turban claims that a slight swelling of the thyroid gland is one of the earliest signs of tuberculosis and is less often seen in late cases. In young women especially when associated with rapid heart beat as often occurs in phthisis it may result in a wrong diagnosis of Graves Disease.

PALPATION.

Although occupying a separate place in the discussion, palpation is usually practised at the same time as inspection is going on; it mostly ~~corroborates~~ corroborates the results of inspection. This is especially the case with regard to the ~~form~~ and movements of the thorax. Special mention may be made here however of Pottenger's ⁽⁴⁾ observations. He laid great stress on the condition of the thoracic muscles in the differential diagnosis between active and latent tuberculosis of the lungs.

The local retractions and lagging movements are he argues, the late results of spasms and resulting degeneration of the muscles over the affected areas. These muscular changes are best detected by lightly going over them with the finger tips; their consistence is an indication of the condition of the underlying structures. All the chest deformities so often met with in phthisis, he argues are due to

these changes in the thoracic muscles.

Apart from the consideration of the thoracic muscles specially, the muscular system generally should be examined, as I hold that often exceedingly valuable evidence is to be obtained from examination of this system. The reason for this is that the muscular system early reveals a characteristic loss of tone which accompanies that run down feeling so often the first thing to be complained of by patients in the early active stage of the disease. There is not the slightest doubt that one of the earliest actions of the T.B. toxin or toxins is to diminish the power of the lower motor neurone, may be by depressing the anterior multipolar cells of the spinal cord. There is evidence of this in the certain amount of atrophy of the muscles which is to be observed in some cases when first examined and it is also evidenced by the loss of weight sometimes complained of. If real early cases are obtained however this atrophy has naturally not had time to set in, but the immediate defect of this toxic depression of the lower motor neurone is at once apparent in that peculiar lack of ^{tone} ~~toxicity~~ which the muscles generally reveal when they are caught up between the fingers of the observer. These as I have said may not be wasted to any appreciable extent but they have a characteristic

plastic feeling, I can describe it in no other way. The nearest gross likeness that I can give to the condition is, that the feeling of the muscles is ~~the~~ like palpating the flesh of a newly killed cattle beast while it is still warm, and before rigör mortis has set in. I have been told that this theory clashes with that of Pottenger who, as I have said holds that there is first a spasm of the muscles overlying the areas of the lesions within the thorax. I do not think it does clash, when in this ~~res~~pect we first consider the researches of Head, and secondly the results of a reflex action from the tuberculous lesions through these sections of the cord then back to the corresponding muscles on the thoracic wall.

The first effect of the lesions, as in many nervous diseases would be irritative, that is we should then have that muscular spasm mentioned, the prolonged effect of this must be to exhaust the spinal cells, rendering these in particular still more liable to the effects of the circulating toxins; finally the muscles in these special areas would show a greater amount of degeneration than those elsewhere.

Corroborative of Pottenger's theory and in contradistinction to the lax state of the muscles generally found elsewhere, I have in many cases been able to feel the muscles

overlying an affected apex in a tense resistant state; this can best be detected by pressing the tips of the fingers firmly down behind the clavicles, any difference on the two sides is at once detected. I am of the opinion that this is sometimes a valuable and early sign in helping to arrive at a diagnosis especially as to whether the lesion is active or latent in the region of an apex. (V. Cases 27 & 29.)

An increased myotatic irribalidity is also indicative of loss of muscular power, a point which Drs. Philip and Gulland used to commonly demonstrate to us by tapping on the pectoral muscles of the patients. There is reason to ~~sur~~ suppose also for much the same reasons as already given that the size of the iden- muscular wave so produced bears some relation to the extent of the underlying disease.

Palpation may reveal any accompaniments to respiration, such as coarse friction or rhonchi. Vocal Fremitus is to be tested for; this is the vibrations produced by the vocal cords. being transmitted through the respiratory apparatus and chest walls to the palpating hand. V.F. is usually increased over lung areas which are consolidated or associated with cavities; it is also increased over that part of the lung lying above a pleural exudation. It is decreased over marked pleural thickenings,

pleuritic effusion, pneumothorax and obstruction to a bronchus by secretion or enlarged glands. It is difficult to detect in fat subjects. Sometimes a tenderness on palpation is to be observed with some patients; I have often noticed this when percussing perhaps rather too forcibly; also when examining the apical muscles as described above.

Palpation will reveal any enlarged glands which may have been missed on inspection; this is especially the case with the supra-clavicular glands. Still ⁽⁵⁾ claims that in some cases of enlarged tubercular glands lying in the posterior mediastinum and upwards, in children, deep palpation behind the manubrium sterni may result in the feeling of one of them. I have often tried this but as yet I cannot say that I have ever succeeded in doing so. Of more value in this condition I think is the loss of elasticity and resistance over the upper part of the front of the chest on pressure, also mentioned by the same observer. ⁽⁶⁾

Chest measurements and the weight should be taken in every case. The healthy relationship between chest circumference and height is as one is to two; if less than one half as it usually is in the so called "Phthisical" chest then the thorax is not to be regarded as a good one. This standard I think in many cases is

often more arbitrary than real.

Of more importance than measurements however, is the amount of expansion got by deep inspiration after forced expiration. In the healthy person this should not be less than $2\frac{1}{2}$ inches. An increase of this expansion is a good sign when occurring in a patients under treatment. The breadth and depth of the chest can be ascertained by the use of calipers. Asymetry may be elicited by moulding strips of lead sheeting to each half of the thorax and comparing the two. The estimation of the vital capacity of the chest by the Spirometer is of value when records of this are kept of a patient under treatment, a gradual increase is of good prognostic value, a decrease denotes the opposite. The sign is of much about the same value as the amount of chest expansion.

I do not think that the comparing of the vital capacity of one with another is of much value, because individual factors play a large part in this. Such are the relationship between the height and the girth of the chest; also practice at the machine has a wonderful effect in increasing the reading. V. Ziemssen ⁽⁴⁾ however claims to have established a definite relationship between the height and the amount of the

vital capacity. He allows 20 ccm of V.C. to every cm. of height, if below this then there is indication of pulmonary disease.

PERCUSSION.

Although percussion is strictly speaking, in part a modified form of palpitation, it differs essentially from the latter, in that with the former the sense of hearing plays a most important role. Two factors come into play, namely; first the detection of the pitch quality and intensity of the notes elicited, and secondly the appreciation by the underlying finger on the chest wall, of the feeling of resistance and a peculiar sense of vibration. A deaf man could not rely on the former factor, yet he need not necessarily be unable to percuss and profit by it, because the feeling of resistance and the vibrations set up would still be left him to appreciate, in his case this sense of perception would be educated to a higher degree than is the case in the majority of those who are possessed of all their special senses. There is no doubt that many fail to gather the full information from percussion because they have neglected the education of the sense of appreciation to this special feeling of resistance or what may be called the "Vibratory Sense". It is not given to everyone to be

possessed of a keen musical ear, yet for the early detection of incipient lung disease, undoubtedly the finger is of more importance than the ear, as far as percussion goes at any rate; the former unlike the sense of hearing is much more a matter of education.

Absolute dullness is not got in early cases, so at the outset it must be understood that when a part of the chest wall is described as being dull what is meant is, that it is relatively dull, or better still, it is of less resonance than normal.

In by far the majority of cases of pulmonary tuberculosis, the disease first commences in the upper lobes, and of these usually in the neighbourhood of the apices, so it is to the apices that one naturally turns first, when commencing to examine a patient by percussion. Somewhere about the summit of the apex in front, may be described as the seat^{of} election for the T.B. It must not be forgotten however that when negative results are obtained here, it is not at all uncommon for the posterior apex to give the first warning. Even failing this one should certainly not pass the care of being free from tubercle. as in not a few cases the first signs of the disease may be shown themselves in the lower lobes, hence the apices of the lower lobes

and also the bases must be carefully examined. The middle lobe of the right lung may occasionally be the first site of mischief, but relatively speaking this is very rare. (See Case XXX)

A knowledge of the normal lung resonance is a "sine qua non" for the detection of incipient phthisis. How is it possible for small changes in the lungs to be detected, if the limits of normal lung resonance are not definitely known? I have not the slightest doubt however that apart from errors in the technique of examination this is one of the most fertile causes of early cases being missed.

With the normal adult, resonance in front descends as far as the VIth rib in the nipple line, to the VIIIth rib in the mid axillary and to the Xth in the scapular line. In the case of left lung an allowance of $\frac{1}{2}$ must be given below these landmarks. For practical purposes it is important to bear in mind, that the front of the chest corresponds with the upper lobe along with, on the right side, a small portion of the middle lobe, whilst the posterior aspect from the spine of the scapula down to the Xth rib corresponds to the lower lobes. As regards the normal resonance in the region of the apex, this will be discussed in the mapping out of "Kronig's Areas".

In health the degree of resonance varies in

in different parts of the chest. Corresponding areas on the ~~hor~~-sides are generally but not everywhere of equal resonance, the latter depend on the relationship of the underlying organs to the eg Heart and Liver. The degree and ~~limbs~~ ^{limits} of resonance vary a little depending upon the stage of respiration. The mapping out of these variations is known as "Tidal Percussion", absence of tidal percussion variation at the posterior apex or base especially the latter is very significant of disease, it may not be tubercle in every case, yet the sign must be looked upon with suspicion.

Interference with this tidal movement along with actual ~~diminution~~ ^{diminution} of air space in the lung, are the two factors bringing about lessening or loss of the lung resonance. With regard to tidal movement at the posterior apex, there is no doubt that this may occasionally be very slight or even absent, and at the same time be compatible with ~~the~~ health. Whether a patient with absence of posterior apical tidal movement however, can be looked upon, as equally resistant to ~~their~~ T.B. as one who has good tidal movement in this region, is quite another matter. Personally when-ever I am consulted by such patients I always insist on ~~their~~ carry-

ing out breathing exercises, if this does not bring about a marked improvement in the tidal variation within a short time I have always a lurking suspicion that the T.B. may be hanging about, quiescent or otherwise, especially if other conditions are also present e.g. in the history or symptoms.

As regards the base, the case is clearer, the absence of tidal movement here, in the majority of cases, indicates pleuritic adhesions, and the practical point to decide is, whether these are associated with active or latent disease.

Topographical percussion of the Apical regions is one of the most important of modern methods in the diagnosis of early lung tubercle.

This method is associated with the name of Krönig⁽⁸⁾ and consists, in the joining by percussion the anterior and posterior areas of apical resonance by an "Isthmus" over the shoulder. It is to be understood, as Goldscheider⁽⁹⁾ pointed out that this is not the same thing as mapping out the upper limit of the lung. What occurs is that the lateral borders of apical resonance are projected as this band over the shoulder. Authorities differ as to the mode of percussing this band, thus Bandelier and Roepké⁽¹⁰⁾ recommend the highest of percussion from within outwards before and behind, Rivière⁽¹¹⁾ however recommends th

highest possible percussion from within outwards or vice versa. My experience is that with some cases, such as those with much fat and muscle heavy percussion is essential. As a rule however most of the subjects met with in hospital or dispensary practice are not such, and so if possible, remembering Dr. Phillips⁽¹²⁾ dictum of always percussing in the direction of less resonance I try, although I must admit that it is not always possible, to avoid heavy percussion. My method is to first roughly find the centre of the band, then to lightly percuss from here outwards marking the limit, next from the same place inwards towards the neck again marking the limit. Having marked out both shoulders the results are compared as regards measurements. Except in cases of deformity the two sides in health should correspond, if they do not it is indicative of a lesion certainly on the side of less resonance, whatever the condition of other lung may be. The lesion is not necessarily due to tubercle, and even if so it does not of necessity indicate active tubercle.

Goldscheider as already stated objected to this method of percussing the apical region on the ground that the apex itself was not mapped out. He set himself to actually percuss out the lung apex in front and behind, so by these means we

have another method of topographical percussion. In front the summit of the apex lies between the heads of the sterno-mastoid attachments, Personally I have given over trying to actually map it out in this region as I never really satisfied myself that I had always done it correctly. Behind it is easier to do, and the normal resonance can be detected almost to the level of the VIIIth cervical vertebra, this depending on the phase of respiration. Tidal percussion about the apex as said before is easiest carried out here. If, after Krönig's areas have been mapped out, the inference is that there is mischief at one or other apex, or may be both, then areas of special dullness should be looked for, (Figs 3&4) and for many of the following remarks with regard to these areas of special dullness I am largely indebted to the lecture of Dr. Lees to the R.C.P. London in 1912. It is over twenty years since Sir James Fowler pointed out from post-mortem evidence that the precise site of the earliest tubercular lesions was not at the actual summit of the lung, but at a place $1\frac{1}{2}$ to 2 below this. He also showed that a second site at the outer part of the upper lobe on the same level is also very common, and that a third early focus is also fairly common about $1\frac{1}{2}$ below the apex of

the lower lobes, a fact which the late Dr. G.A. Gibson used to impress upon his students. Now it is quite possible to detect these areas by percussion, and for this the best attitude for the patient to be in, is lying evenly and easily in bed on a firm mattress. It then becomes fairly easy to be detected dullness in the first and second spaces, corresponding to the diseased areas demonstrated P.M. by Sir James Fowler and others. A point to remember for practical purposes is that $1\frac{1}{2}$ of the collapsed being of the cadaver, corresponds to about 2 of the air containing lung during life (Lees) The second and third spaces must also be examined, as well also as the remaining part of the anterior wall of the chest along with the axillary regions.

For the examination of the posterior aspect of the thorax it is advisable that the patient should be sitting up with his back to the examiner, he should bend slightly forward with the arms crossed over the front of the chest.

Percussion over the whole of the supra-scapular fossae should be practised as mentioned above, in the mapping out of Krönigs Areas. In the case of incipient phthisis a dull area will often be found in this region quite close to

the first and second dorsal vertebrae corresponding anteriorly to a dull area at the inner end of the first intercostal spaces. In like manner a dull area may be found at the outer part of the first interspace, and often a fairly large area in the region of the lower angle of the scapula corresponding to the upper part of the lower lobe of the lung.

The areas mapped out in front may possibly extend down into the second interspaces, and in more severe cases the outer areas may extend into the axillary regions. There are thus six dull areas to be specially looked for when examining a patient for the first time, namely two in each upper lobe and one in each lower lobe. They are probably the most important and at the same time the most easily detected part of the physical signs in a case of early pulmonary tuberculosis. They do not by any means represent the complete picture but they are sufficient for diagnostic purposes. My experience like that of Dr. Lees is that they are always present to a greater or less degree in almost every case except in those few cases where attacks of pleurisy have been probably fairly frequent before a definite examination has been possible. The important question however is whether these areas in all cases can be accepted as

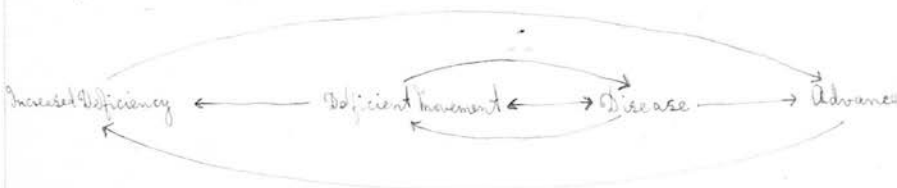
definite evidence of tuberculosis? In the case of adults the answer is probably "Yes" with regard to children I am not in a position to give an opinion. The diagnosis of pulmonary tuberculosis in children is always a difficult matter under the best of circumstances, especially when one remembers that the lobular areas of collapse are not at all uncommon in feeble children. However in these cases, if definite areas of dullness are associated with enlarged *bronchial* glands, which I think are often within the bounds of fairly easy diagnosis I always feel justified at present in my own mind at any rate of regarding the case as one of pulmonary tuberculosis. Ofcourse it is always to be understood that the discovery of any of these dull areas does not neccessarily mean active tuberculosis, or as Prof. Von. Behring ⁽³⁾ has put it "Tubercular infection does not neccessarily mean tubercular consumption". They may be the only recognisable reminders left of a healed lesion, because there is no doubt that when once formed they may shrink, but never entirely disappear.

The question whether the disease is active or latent can only be answered by taking into consideration all the evidence obtainable from

every possible source, no claim is or can be made that percussion settles this point. What is important however is that an indication of the progress of a patient under treatment may be got by careful observation of these areas if associated with active disease. When shrinking along with lessening of the auscultatory phenomena and improvement of the condition generally is observed, the prognosis is eminently favourable. This ofcourse can only be gathered by keeping the patient under extended observation, with frequent examinations at intervals of certainly not greater than one month at first. For this again, it is most important that diagrammatic records be kept. Mention may be made of other dull areas which may often be detected, but which in early cases are not nearly so frequent as those just described.

On the anterior wall a small area may be discovered in the third intercostal space just above and interval to the nipple, further out again another area in the same space may be found just below the anterior fold of the axilla. On the right side corresponding to the middle lobe of the right lung, a definite area may be found just internal to and below the nipple. Of these dull areas it is seen that they

are most common in the apical regions and not necessarily the apices of the upper lobes. The apices are least expanded on respiration, and if a tubercle commences here, especially if it also involves the pleura, a still further arrest of the local respiratory movements occurs, as mentioned before. This is probably of a reflex nature analogous to the inhibition of the diaphragm movement which occurs in cases of local peritonitis. This increased failure in expansion makes the local conditions still more favourable for the further advance of the disease, and so we have another example of a vicious circle.



Thus the disease advances more quickly in the apical regions than elsewhere, and hence it is that caseous areas and cavities are far more frequently discovered P.M. in the region of the apices than in other parts.

Before finishing the consideration of percussion mention may be made about some special varieties of resonance that may be elicited.

"**TYMPANITIC**" resonance is the term applied to the note elicited when percussing over cavities

containing air, or over the lung, the contained air in which has become diminished in tension. It is heard over a ^{new} premothorax and may then be called "Amphoric" because of the musical character which has been imparted to the note.

Another form of tympanitic resonance is the "Skodaic", elicited from over that part of a lung lying above a pleural effusion, or over the upper part of a lung the lower part of which is consolidated e.g. by pneumonia, the air in these cases is relaxed in tension resulting in the tympanitic character being given to the percussion note.

Cavities within the lung substance may give rise to tympany on percussion but I cannot say that I have ever been able to diagnose a cavity from percussion alone.

Williams Tracheal Note. is the tympanitic resonance produced when there is consolidation of the lung substance between the chest wall and the trachee or the primary bronchi, this may give the impression of a cavity.

Cracked Pot Resonance is also partly tympanitic in character and is obtained on percussion over a cavity in connection with a large bronchus; the contained air within the cavity is forced through the constricted orifice into the bronchus the result is a clinking sound.

Wintrich's Signs is produced when the tympanitic

 resonance is higher with the mouth open than
 when it is closed. The sign probably suggests
 a cavity partly filled with flind.

Gerhardt's Sign depends on the flind present

 in a cavity altering the note on change of
 posture, it also suggests a cavity (Oval?) part
 -ly filled with flind, it is best detected over
 a pneumothorax.

Trousseau's Phenomenon or the Bell or Anvil

Sign is got when auscultating over a cavity

 and at the same time percussing with two coins
 instead of the fingers, a sound like the ring-
 ing of a bell or a hammer striking an anvil is
 heard through the stethoscope. The sign is best
 got with a pneumothorax.

Freidreich's Sign consists in the tympanitic

 note becoming higher or even disappearing on
 deep inspiration, owing to the increased tension
 produced in the air within parts of the affect-
 ed lung.

Finally it must be said that examination by
 percussion is never complete until the areas of
 superficial and deep dullness of the heart
 have been mapped out, invaluable information
 may be gained from this. Thus if a pulmonary
 lesion is associated with much contraction

displacement of the heart

displacement of the heart to the affected side is at once noticed, even if no actual displacement occurs an increase of the dullness to the right or left may be noticeable owing to the covering parts of the lungs receding from over the borders of the heart, leaving it as it were more exposed. A most characteristic sign of old right sided disease Dr. Phillip used to point out is apparent increase of the heart's dullness to the right, displacement may not be present. Again with relatively great fibrosis of one lung compensatory emphysema may result in the other, resulting in a smaller area of superficial cardiac dullness. If both lungs are fibrosed then this area tends to become increased in size .

ANSCULTATION.

It is commonly supposed and stated that the earliest and therefore most important indications of the presence of pulmonary tuberculosis are revealed by the stethoscope. I may be mistaken or I may like others be heterodox, but as yet I am by no means convinced of the truth of this, or at present likely to be so. Ofcourse it may be well at the outset to request those who hold to this view to define what they mean by "The most important indications". If it is

argued that most is learnt about the condition of the lungs in an advancing active case by this means, and that these signs are conspicuous above all others, then in that sense it may be granted. If however, taking any ordinary case from its ~~onset~~ ^{onset} and the truth for the statement then claimed I venture to contradict it. Again if the statement is to be accepted for diagnostic purposes in general, what striking success may I ask is to obtain in the so called world wide crusade against tuberculosis that is supposed to be going on a present? It is like saying that the most important indication of a horse being stolen is the finding of the stable door open and no horse. I would rather say that the most important indication here was the catching of the thief as he was about to open the stable door especially if one had suspicions that there was a thief in the neighbourhood.

The consideration of ^{an} auscultation comes under the following heads:-

1. Type of Respiration.
2. Modifications of Type.
3. Accompaniments.
4. Vocal Resonance.

Personally, after examination by percussion, and especially if areas of dullness have been found I always proceed to the testing of vocal

resonance first.

VOCAL RESONANCE like vocal fremitus in the normal

chest, is greater over certain areas than over others, this must be understood at once or mistakes may occur. It is normally relatively greater over the right apical region than over the left, it is also relatively increased behind over the region of the roots of the lungs. The normal anatomy of the chest explains the reasons of this.

"BRONCOPHONY" is the term applied to the V.R. when there is marked increase, the sound is just as if the voice was spoken into the stethoscope, this is normally got when auscultating over the trachea. In pathological conditions broncophony is heard over lung consolidations, and has the same significance as bronchial or tubular breathing, with this difference however that broncophony may be recognised with a lesser degree of consolidation than definite variations in the type of breathing may be.

"PECTORILOQUY" is the term used when the V.R.

is very markedly increased, so that even the whispering voice may be distinctly heard. It is also normally heard over the trachea or larynx, but pathologically it is usually associated with a fairly large cavity communicating with a bronchus. It is also partly obtained

occasionally when auscultating over the upper part of the lung, when the lower part is compressed by an effusion.

Decrease of V.R. is relatively and normally the case over the left apex, pathologically a decrease is associated with emphysema, whilst over the lower parts of a pleural effusion it is absent altogether, it is much diminished or even absent over dense pleuritic thickenings.

Special varieties of V.R. are spoken of as

Amphoric, Aegophonic and Cavernous.

AMPHORIC or Echoing V.R. has a metallic ringing sound associated with it, and is heard best over an open pneumothorax and occasionally over specially large vomicae in the lungs.

AEGOPHONY or Punchinello Voice is a modified form of V.R. heard over the upper layer of a pleural effusion; the resonance here has a peculiar nasal twang about it, supposed to be like the bleating of a goat, hence the name. Stone⁽¹⁴⁾ considered that this form of resonance was due to the interception of the fundamental tone to a greater degree than the harmonics or overtones. Prof. Wyllie used to teach us that it was the other way about i.e. the overtones were intercepted to a greater degree than the fundamental.

CAVERNOUS resonance is a more or less intensified

broncophony, it does not however possess the ^{same} ~~same~~ high-pitched character as the latter.

It is not an important sign and its association is supposed to be with a lung cavity surrounded by a certain amount of fibrosed or indurated lung tissue.

Trousseau's Bell phenomenon has already been discussed under percussion.

HIPPOCRATIC SUCCUSION although not belonging to

V.R. may be noted here. It is the splashing sound which is heard when a patient suffering from a pyo-pneumothorax is gently shaken, I do not think it is a justifiable test to carry out at any time, as the condition is more easily diagnosed in other ways and certainly in a much more comfortable and less dangerous way for the patient.

RESPIRATION IN PHTHISIS The quality and strength

of the sounds and their relation to inspiration and expiration must be carefully noted.

The first abnormality be noted in an early case may be a simple ^{or} ~~weaking~~ ^{weakening} in the intensity of the respiratory murmurs, which may or may not be of an interrupted character, almost amounting sometimes to Cog-Wheel type. This, according to many authorities is due to the heart pulling on the lung tissue, others again ascribe it as due to the presence of swelling of the

lining of the finer air passages, or as due to small rhonchi which have not yet become accompanied by secretion (Sahli) ⁽⁵⁾ This type of breath sound may again be accompanied by harsh vesicular breathing, and indeed a harsh vesicular breath sound may be the only change to be noted in an early case. If these changes are limited to the apex of one lung only, then the greater is the likelihood of tubercle being the cause of the conditions. Harsh vesicular breathing even if also cog-wheel in type heard over both apices may mean nothing, in this neighbourhood such an occurrence is quite common amongst coal miners and I have often thought that pneumoconiosis was the cause of this, especially as the occurrence is more frequent amongst older pitmen than younger.

These exaggerated local breath sounds however may be due to a commencing fibrosis around small tubercles. Weak breathing over a large area is probably due to pleuritic thickening over the part, or to the partial compression or obstruction to a large bronchus. In many cases the characteristic "Clicking" or mucous râle may be heard, the explanation of this depends on the passage of the contained air through moist tubules. Such râles are easier detected than changes in the type of breathing only, yet they

are frequently missed owing to incomplete methods of examination. To elicit them if present, the patient should be asked to make one or two sharp coughs immediately before ~~insp~~ ^{inspiring}, they may then be heard after this when otherwise they might have been missed. In some cases a positive diagnosis may be made by this sign alone when other methods have failed to give any or sufficient direct evidence. With advancing disease the type of breath sounds becomes more bronchial or tubular in character, this is owing to the consolidation present conducting the sound better from the bronchi to the surface. Often however there are intermediate stages between weak or harsh breathing and definite tubular breathing, these are known as Broncho-Vesicular or Vesiculo-Bronchial sounds according as the bronchial or the vesicular element predominates. Sometimes however these forms are spoken of as "Indeterminate" breath

 sounds.

While these changes are going on there is usually at the same time a lengthening of the audible sound on expiration, especially is this the case if areas of emphysema are also present. As a rule recognisable changes occur sooner in the inspiratory phase than in the expiratory. When definite bronchial breathing occurs then

the expiratory sound is much prolonged, and is often more bronchial in character than in the inspiratory phase.

It must be remembered that all these changes in the breath sounds are often much clearer over the right apex than over the left. The explanation of this lies in the normal anatomy of the parts. On the right side the bronchus is wider and straighter than on the left.

Throughout all the changes in the type of breathing, the mucous râles mentioned above may or may not be present. Their presence all depends on whether catarrhal processes are present or not, and these are certainly not essential in incipient cases. It is quite common in slowly advancing cases for changes to be noted on percussion and in the character of the breath sounds without the presence of râles. These are precisely the class of cases that give rise to difficulty in early diagnosis and which usually advance slowly and insidiously; this is therefore the reason for my argument that percussion is often of much more value in the diagnosis of doubtful cases than auscultation. The recognition of râles however is an important sign not only for diagnosis in many cases, but in estimating the activity of the disease. More quickly advancing cases are sooner accompanied

by catarrh than the slower chronic fibroid cases but the former cases however are more easily and usually sooner diagnosable. It is another point depending on the virulence of the seed and the fertility of the soil.

RÂLES are described as numerous, fairly numerous

or scanty according to their quality, as coarse medium or fine, according to their size, as metallic, tinkling or consonant according to their character, and as moist or dry according to their sound.

Dry râles are usually called "Rhonchi" which again ^{may} be described as crackling, whistling or sonorous according to their variety, their names speak for themselves. Rhonchi are supposed to be due to the presence within the lumen of the bronchioles, of masses of viscid tenacious secretion, hence their temporary disappearance partially or wholly after a bout of coughing. Examination of the chest by ^u auscultation should be after the same manner as percussion i.e. special attention should be paid to those areas which are likely to be the seats of early disease. All the morbid ^b sounds mentioned above become much more marked in the presence of a cavity. The bronchial breathing may become amphoric or cavernous in character, the crepitations become ^w loud coarse and exceedingly numer

-ous, if at the same time these changes are accompanied by Wintrich's or Gerhaddt's percussion signs the diagnosis of a cavity is certain.

Before concluding the consideration of auscultation, mention may be made of D'Espine's ⁽⁶⁾ method of spinal Auscultation of the voice in the diagnosis of Enlarged Bronchial Glands in children.

The child is made to repeat the number 99 while the examiner auscultates over each spinous process in turn. Over the cervical spines the voice is clearly heard and has a characteristic clear tracheal quality. Normally this quality ceases opposite the Vllth spinal process at the level of the pulmonary apex. If enlarged bronchial glands are present however the alteration in the voice does not occur until well below this level, in very marked cases the bronchophony may extend as far as the Vth dorsal spine. The bronchial quality is then due to enlarged glands which at this level surround the trachea and its bifurcation and extend backwards towards the vertebral column and so conducting the sound directly between the two. As age advances the level of the bifurcation of the trachea sinks, in children it is opposite the lll D.V., later it reaches the level of

30

of IV D.V. whilst in old age it may be as low as V or VI D. V.

(14) Zabel gives the following levels, below which in children spinal bronchophony is suspicious.

Young Children Vll C.V.

About the Eight Year I D.V.

About Fifteenth Year lll D.V.

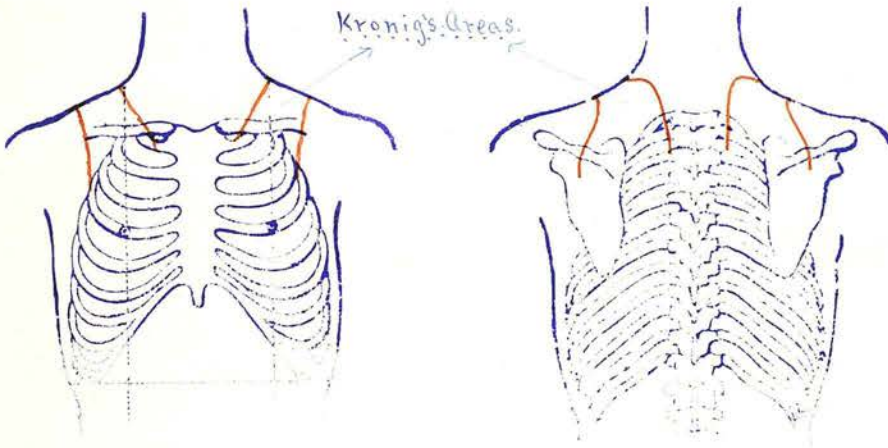
Immediate ^uauscultation reveals the bronchophony more distinctly than the stethoscope, but the latter enables more exact delimitation. Lately however I have been trying the vibrating ~~tuning~~ tuning fork placed on the trachea immediately below the larynx, and then auscultating down the spinal processes. I think this method gives the most exact delimitations, the sound of the fork rapidly tailing off as soon as the conducting areas are passed. It was Dr. Cautlie's article (15) on the value of the tuning fork in auscultation which appeared in the B.M.J. a few months ago, that first suggested to me that this might be of value in the diagnosis of Enlarged Bronchial Glands in Children and which I have always looked upon as a very difficult matter in many cases. As far as my limited experience goes I am of the distinct opinion that the tuning fork is a splendid aid. (V. also The Diagnosis in Children p.)

The symptoms and physical signs having now been discussed, it is my intention now to proceed to the consideration of the role that tuberculin plays in the diagnosis of phthisis pulmonalis. It might be argued that the consideration of the bacteriology of the disease should take precedence to this, as after the consideration of the physical signs this would be more logical. This ofcourse cannot be denied but as part of the thesis is to be given up to the discussion of Laboratory Methods, the question of the bacteriology will find a more suitable place there, if only convenience of arrangement may be put forward as the excuse for this.

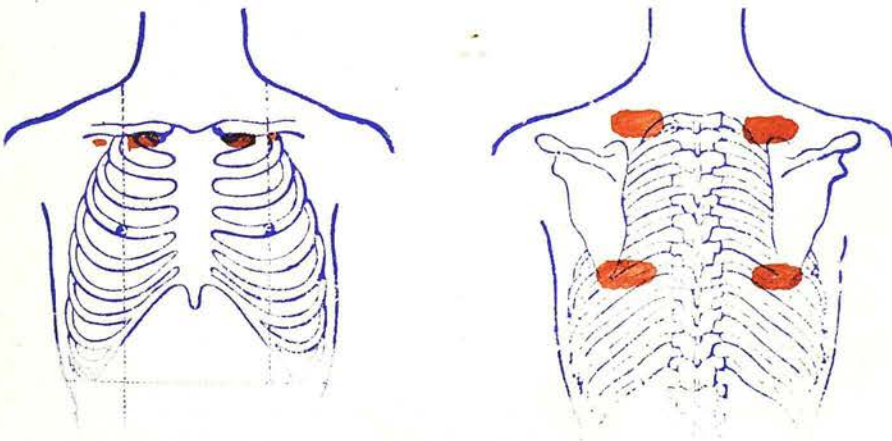
Tuberculin some might say, should also come under the same part, as it is essentially a product of the laboratory. Whilst this also cannot be denied my excuse in this case is that it is not the intention of this thesis to take up the consideration of the preparation of tuberculin and its derivatives as this may be found in numerous pamphlets issued to members of the medical profession by many of the more or less interested firms.

For diagnostic purposes Koch's Old Tuberculin or Tuberculin-Alt is almost exclusively used, so taking for granted its mode of preparation there is certainly no need to defer its consider-

ation until later.



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TUBERCULIN IN DIAGNOSIS.

"And wilt thou mix Hellebore who doth not know
How many grains to the mixture go?

The art of medicine this forbids I trow!"
(Sir Walter Scott.)

In medicine history there is probably no medicinal agent that has had a more romantic career if it may be so termed than the Tuberculin of Koch. To be given to the world by its author in a state of premature birth, for great hopes to be aroused by it as pauacea at last discovered, to be like the prophets of old stoned and cast from without the fold before its uses were definitely settled, for the maturer knowledge of the faithful few to lead it back into the fold again, this time to receive fairer consideration, to be now proclaimed as one of the great discoveries of modern medicine, all this is a matter of recent history and is rapidly being forgotten, what the future of tuberculin and its offsprings may be, no one dare yet prophesy. What medico is there who will deny the unfailing power of tuberculin to make the lurking bacillus declare itself?, it is perhaps too powerful in this respect in most cases. True it has its limitations in that it is not always possible to discriminate between the tuberculous and tuberculised. Can it be denied however

that in spite of this, a mighty weapon has been put into the hands of the clinician? so mighty indeed that even at the present day its own or its offsprings powers are far from being completely recognised. With regard to the use of them as therapeutic agents a titanic controversy is so raging at present among our authorities, that the humble and cautious general practitioner ever bearing in mind "Levit^{us} est inanem aucupare rumorem", does not know as said before where to turn his head for light. Observations and denials, along with so much of what is probably mere speculation, are so intimately mixed up in this vexed question of tuberculin in treatment, whilst at the same time one feels that one's own experience in this respect has been so limited that I am almost afraid to express an opinion. I am of the belief however that tuberculin is of great value in certain selected cases and that it is to this agent or to its progeny, that one will have to look for a specific remedy for tuberculosis if ever such an agent is discovered. When one comes to consider the use of tuberculin and by this I mean Koch's old tuberculin, in diagnosis, it must be understood from the very beginning that it is only rarely that the agent is required. If the diagnosis can be made by

ordinary clinical methods, ~~which~~ need is there for tuberculin in this respect? A medical man who for this purpose confesses to the frequent use of tuberculin, at the same time whether he is aware of it or not, also confesses to his own lack of clinical knowledge. It is only rarely that one meets ~~with~~ cases, and I mean here really active cases and not old healed lesions, which are not recognisable by subjective and objective clinical data. When the exceptional and obscure case however does ~~him~~ up, it cannot be denied that tuberculin is a most valuable agent. Tubercular sensitiveness or 'Allergie' is the condition which tubercular persons possess, and which renders them extremely ^{susceptible} to the products of the tubercle bacillus, which in the case of the healthy, have little or no effect.

Many theories have been advanced to account for this condition of sensitiveness, the discussion of which however is quite outside the scope of this thesis. A working hypothesis nevertheless is essential, and for this, the theory of Woelff Eisner ^① ["Handbuch der Serumtherapie" (Munich 1910)] is I think, the ~~simplest~~ simplest and most satisfactory. This observer assumes that the tubercle-infected person has produced in his body specific anti-bodies which

act by breaking down the tuberculin molecule and so allows of the setting free of toxic products. These anti-bodies are known as "Lysins." This theory will explain all the characteristic features that have been observed with regard to injections of tuberculin into different persons. In the case of the healthy or non-infected person no Lysin is present, hence tuberculin becomes a harmless circulating foreign body which is quickly eliminated. In the case of infected persons the action is very different. Here the Lysins present break up the tuberculin at the site of injection with the production of a local reaction, if sufficient has been injected for some to reach the circulating blood then there is also a general reaction, as well also as a local reaction at the sight of the lesion.

The result of these injections is probably an increase in the number of the anti-bodies present in the blood, a most important point when one comes to consider the question of the Opsonic Index and the production of Anti-tuberculin.

It may be accepted as a fact that nowadays tuberculin has become in the hands of the careful clinician a harmless yet valuable agent. Danger is the result of careless and indiscriminate administration. Correct conclusions can only follow on rigid precautionary methods

being adopted, the reaction being of great or little value according to the employment or otherwise of certain well established principles For the diagnostic purposes the tuberculin tests are classified as follows:-

- (1) Subcutaneous.
- (2) Cutaneous.
- (3) Percutaneous.
- (4) Intracutaneous.
- (5) Ophthalmic.

THE SUBCUTANEOUS METHOD.

The basis of this test is the production of a general reaction, marked by increase of temperature and general constitutional symptoms, as well as occasional local manifestations. At first, objection to this test was raised on the ground that a positive reaction occasionally occurred in apparently healthy non-tuberculous ^{subjects}.

The explanation of this probably ^{is} that these persons were the subjects of old healed lesions i. e. "Tuberculised" or that class of person referred to by Dr. Philip in his address to the International Congress of Tuberculosis at Rome in 1912. ~~Reported in~~ This very fact detracts to some extent from the value of the reaction because latent tuberculosis in some form or other is practically universal amongst civilised communities.

It is useless to try this test on a patient

who is the subject of pynexia, a fever of even 99 may be enough to prohibit its use. The reason for this is because one of the results of a tuberculin reaction is the raising of the temperature from one to three or four degrees. It is thus shown how necessary it is to keep a careful four hourly record of the temperature for almost a week before the test is attempted. In this way the fluctuation following an injection may at once be distinguished from any ordinary fluctuations the individual may have. The reaction usually sets in about the 10th or 12th hour after injection, occasionally however if it may be delayed until the end of 24 hours. The constitutional disturbance is marked by a ^{rise} use of temperature, feeling of chill, headache, general malaise, pains in the back and limbs, and may be a feeling of sickness or actual vomiting may occur. These symptoms soon pass away however. The most important part of the test when employed for the diagnosis of phthisis pulmonalis is the occurrence of a focal reaction at the seat of lesion of the lungs, it was precisely due to this that tuberculin was so vilely execrated by many soon after its introduction into this country. This is also the reason why it is so dangerous a test for the careless or unwary to adopt. The truth is that in these cases a larger dose has been

given than the circumstances warranted.

With regard to dosage there is still some difference of opinion as to the amount to be used for the first injection. Some start with an initial dose of .1 milligrams, others again start right off with a dose of 2 or even 3 milligrams. Personally I think that if the reaction can be sufficiently well obtained with a small dose there is nothing gained by using the larger, there is certainly more risk and more discomfort to the patient with the larger. Should this first dose fail to produce a reaction a second dose of .4 or .5 milligrams should be given after an interval of not less than one week, the temperature being kept under strict observation as before during this interval. Further doses of .5 or .6 milligrams may in like manner be resorted to if necessary. If no reaction is still the result the case may be looked upon as non-tuberculous. It is very rarely that the large doses are required in order to confirm a diagnosis. If the reaction after the first dose should be doubtful the same dose should be repeated after 3 days. The objection to the repetition of small doses is that "Tolerance" may result, so that the reaction may not be obtained even with larger doses. This principle is well known to some cattle dealers who often so dope their cattle before

sending them to the market, and in this way allow of their passing the tuberculin test which some dairy farmers insist upon before taking over the cows.

A reaction is considered as positive when the general symptoms related above occur wholly or in part and at the same time the temperature rises at least one degree (See Case X X)

Ocasionally at the site of injection a local reaction is to be observed in the occurrence of redness and induration the needle track reaction of Escherich. The dilutions for the injections should always be freshly prepared as marked deterioration soon occurs after 48 hours

A knowledge of the contraindications to the use of the test are essential. The chief ones are as follows:—

- (1) Any degree of pyrexia.
- (2) When the diagnosis is otherwise certain.
- (3) Recent haenophysis.
- (4) Advanced heart disease.
- (5) Acute and Chronic Bright's Disease.
- (6) Epilepsy (Here convulsions may occur even after being dormant for years)
- (7) Hysteria (When oscillations of temperature occur which may confuse the issue)
- (8) General Conditions e. g. Intestinal Tuberculosis (suspected). Immediately after severe illnesses like Typhoid, Scarlet, Pneumonia.

Severe Diabetes. High blood pressure.

CUTANEOUS METHODS.

This is often called the Von Pirquet⁽⁴⁾ reaction because he was the first to apply in a practical manner observations, which had been made both by himself and others regarding the local conditions produced at the site of injection after the subcutaneous method. The method is, after first cleansing the skin, to apply a drop of old tuberculin to the skin over the front of the forearm for convenience, and then to make an abrasion through the drop. An ordinary vaccination instrument does very well for performing the test, care must be taken however to avoid bleeding, as if so, the reaction may be obscured. Three or four sites on the forearm are inoculated, two through tuberculin and the other or others through normal saline, the latter act as controls. If a positive reaction occurs, at the end of twelve hours marked hyperaemia sets in over the sites of the tuberculin inoculation. This hyperaemia gradually increases to reach a maximum at about the end of 24 hours, it may rarely be delayed for another 10 or 12 hours beyond this. Rarely the reaction may set in after 3 or 4 days and then last an equal time before disappearing. The inoculated areas are not only inflamed looking, but also swollen

and hard to the touch.

PERCUTANEOUS TEST OF MORO.⁽⁵⁾

This is a modified form of the above. The tuberculin is rubbed into the skin of the patient in the form of an ointment consisting of 10cc of old tuberculin in 10 grains of lanoline. There is nothing very special about the test. A positive reaction is marked by the appearance of a papular rash over the site of inunction.

INTRACUTANEOUS METHOD OF MANTOUX.

This is practically only a modified form of Von Pirquet. The tuberculin dilution containing about .01 milligrams is injected into the superficial layers of the skin. A positive reaction occurs in from 8 to 10 hours in the track of the needle. There is a central nodule surrounded by a bright areola. The nodule disappears after 3 or 4 days.

OPHTHALMIC REACTION OF CALMETTE OR WOLFF EISNER.

A drop or two of a sterile solution of tuberculin is instilled into the conjunctiva. Calmette recommended a solution made from tuberculin which had been precipitated with alcohol, he thought the glycerine present in ordinary solution of tuberculin might irritate the eye and so confuse the reaction. It is very doubtful however whether the amount of glycerine present

in a 1% solution of tuberculin which is used for this reaction could produce any appreciable irritation of the conjunctiva.

The positive reaction is shown by a varying amount of injection of the conjunctiva. It may vary between slight redness of the caruncle only ^{to} and an intense hyperaemia ^{of} the whole conjunctiva at the same time there is usually a varying amount of fibrinous or fibro-purulent exudation. The reaction may be recognised after 8 hours, it increases to a maximum after about 12 hours and begins to subside after about 24 hours.

Delayed reaction may take place after 48 hours and may then be very severe. A contraindication to the test is the presence of any ocular disease especially if this so, if the disease is tuberculous. The possible danger of the test is the production of serious damage to the eye which has been reported on several occasions.

Personally on the occasions on which I have used the test I have never seen any untoward

result of a positive reaction, certainly however a severe reaction is most unpleasant to the patient, If the eyes are healthy and a careful examination is made beforehand to ascertain this there is probably very little danger to be anticipated in the performing of the test, nevertheless in the light of published cases from certainly experienced observers the

possibility of damage cannot be denied, hence the test should not be performed indiscriminately on all occasions.

LATER MODIFICATIONS OF THE ABOVE TESTS.

Ellerman and Erlandsen⁽⁶⁾ and others have modified Von Pirquets test by applying tuberculin in varying degrees of concentration over different parts of the patients forearm. The point is to find the dilution which just succeeds in producing a reaction. The reciprocal of the fractional dilution which attains this is usually as the indicator of the patients sensitiveness. Ellerman and Erlandsen put a 1% concentration or a sensitiveness figure of 100 as the limit beyond which active disease may be assumed, this however is purely arbitrary.

THE DIFFERENTIAL CUTANEOUS METHOD OF DETRE.⁽⁷⁾

is another modification of the Von Pirquet and consists in applying to different parts of the patient's skin the following.

1. Human tuberculin.
2. Bovine tuberculin.
3. Filtrate of culture of human T. B.
4. Filtrate of culture of bovine T. B.

An attempt is made in this way to distinguish between the various reactions with a view to ascertaining the best form of tuberculin to

use in treatment. It has not been proved however that actual differentiation has been established, nor that the alleged human or bovine type is really present in the individual concerned.

Carle Woodcock's modification is to apply the tuberculin to a denuded surface produced by the application of a small blister. A control surface is also prepared for comparison.

Personally I do not think there is much to be gained by this procedure because nowadays the aim of cutaneous tuberculin tests is rather to observe with what ease they are produced, and not as in this method to do everything possible to obtain a reaction, there is nothing to be gained for the individual case by trying to prove that almost every person will react.

COMPARATIVE VALUE OF THE TESTS.

Undoubtedly the nature of the reactions in all cases must be regarded as specific. A positive reaction must be accepted as strongly indicating the presumption that there is tuberculous infection in some part of the patient's body. A negative reaction means nothing if clinical evidence clearly demonstrates the presence of tubercle, in fact the reaction is usually negative in advanced or rapid advancing cases. The reaction is usually most marked in early

or unsuspected cases, hence it is that sensitive-
-ness to tuberculin must be looked upon as a
favourable sign in the treatment of tuberculosis
a point which is apt to be forgotten or not
recognised by many who use tuberculin in treat-
ment. The meaning of this sensitivness is
probably that the blood contains an unusually
large amount of "Lysins" hence a fair amount
of natural resistance to the disease.

A most important difference between the cutaneous
and ophthalmic tests is, ^{that} the former is much
more frequently positive amongst adults than
amongst children, this probably means that
amongst adults there is a large amount of old
healed tubercular lesions. The inference is
that the cutaneous test is of comparatively
greater value in the case of children than in
adults, and this is vicē versā in the case
of the ophthalmic test. An important bearing
in this particular is probably that the con-
junctiva is a much more absorptive surface than
the skin.

Von Pirquet ⁽⁸⁾ has noted by careful observations
that the percentage of cutaneous reactions
corresponds very closely with the prevalence
of tuberculosis amongst children as revealed P.M.,
this has been confirmed by numerous other
observers. He has also shown that during an
attack of measles tuberculous children have not

responded to the reaction, this he attributes to destruction of the specific anti-bodies in the blood during measles. Clinical evidence bears this out as tuberculosis frequently advances rapidly after an attack of measles.

With regard to tuberculous infants on the breast, tests have been applied by Petruschky with a negative result in all cases, this is probably due to the extreme virulence of the disease amongst infants when it does occur.

From 6 to 12 months 50% reacted, from 7 to 14 years 75%, from 15 to 20 years 87%, from 20 to 30 years 87%, and from 31 to 40 years 87%, From 40 to 76 years 81%. All these cases showed definite clinical evidence of the disease or exhibited very suspicious signs.

About two years ago I tested the Von Pirquet reaction on a number of out patients at the Monkwearmouth Hospital, I found that apart from any clinical evidence as to the presence or absence of tuberculosis about 4 in every 5 adults showed a positive reaction in some form (See Appendix.)

It is evident however that exact conclusions as to the precise diagnostic value of the cutaneous reaction in most cases can hardly be estimated, the reason of this is that the very delicacy of the tests vibrates against

them. Positive reactions certainly indicate the presence of tubercle whether active or latent. Nowadays I seldom resort to the Von Pirquet reaction for any help in diagnosis, but where I meet an early case of phthisis as diagnosed from clinical and other methods I often resort to the Von Pirquet for evidence of prognostic value. With such cases I discard a negative result but look upon a marked positive reaction as of some value in indicating the likelihood of a favourable prognosis under proper treatment.

What is wanted are some simple tests which will demonstrate early active tubercle and not latent; the latter for all practical purposes can be looked upon as cured tuberculosis, spontaneously so or otherwise, and which because of the practically universality amongst the civilised peoples strongly militates against the value of these cutaneous reactions. An important ^{point} however is that, with the exceptions noted above, a negative result probably definitely excludes tuberculosis, the importance of this is minimised however, at any rate in my own experience, in that a truly negative result in any suspicious case is very rarely to be obtained. However it is possible that there may be cases where such a negative

result may be of value as an eliminative aid to the diagnosis of the underlying condition. With early active and favourable cases as said before, a brisk reaction is usually got with small doses indicating considerable resistance on the part of the patient. In the case of old quiescent lesions probably a larger dose is required to get a marked positive result and probably also the reaction is delayed. The so-called focal reaction is a most important indication when obtained, of the presence of active disease. My own opinion is however is that it is very seldom justifiable to resort to this as an aid. In the case of pulmonary tuberculosis this focal reaction is evidenced either in the production of more definite symptoms such as cough pleuritic pain and greater variations in the temperature, or in an increase of the physical signs over the suspected lesions e. g. definite crepitations may now be heard. Such a sequence of symptoms is often seen in cases showing how they ought not to be treated by tuberculin.

The subcutaneous test although attended with greater risks is certainly the most delicate and most useful when wanted for the purpose of diagnosis, as from it the most information is to be gleaned, although I have to repeat

with almost wearying reiteration that here again to be of any use, it cannot be divorced from the clinical evidence, before a definite diagnosis of early active pulmonary tuberculosis can be arrived at.

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LABORATORY METHODS.

"Great is Science and it will prevail".

(Sir W. Turner).

Many have held that until the laboratory has issued its verdict, a definite diagnosis of "Consumption" cannot be made. If the verdict is a positive one indicating that T.B. have been found, then if any doubts existed before, they must vanish now; the proof is absolute. The converse does not hold good however; the absence or rather the non-finding of T.B. does not negative a diagnosis of tuberculosis, yet I am afraid that many patients have lost their only **chance of recovery** because forthwith their sputum having been examined it was reported to have not contained T.B. The wording of many reports has been and still is, unfortunate and apt to mislead some of our elder brothers in general practice.

A negative result means very little, and if there should be any clinical signs at all no value should be attached to it, especially is this so from a treatment point of view if, because of this report, delay is to occur.

In the present light of our knowledge I can confidently challenge any bacteriologist to state definitely, from the results of microscopic examination alone, that T.B. are absent in a given specimen of

sputum; the most he can state is that he never found any, a very different thing indeed to many of the reports bacteriologists send, or used to send, out.

Until a comparatively short time ago the Ziehl-Neelsen method of staining was the only method used for microscopically demonstrating T.B. More recently, as Uhlenhuth, Much and others have shown, there are hundreds of cases where the T.B. can be demonstrated in a sputum when the Ziehl-Neelsen method totally fails to achieve this. Even with these failing to produce evidence, animal inoculation may still prove their presence in many cases. Enough has been said here however to show what little value can be put on a negative report from the examination of a single specimen of the sputum; how criminal then it becomes to withhold proper treatment on these grounds if the clinical signs in themselves alone should suggest it.

As previously stated the finding of T.B. in the sputum is proof positive of the presence of a tuberculous lesion. This does not mean however that the stage of the disease can be inferred from this, other-wise than, that the disease is active. It means that somewhere in the lung or its connections some tuberculous focus is in communication with a bronchus, and on the degree of freedom of communication between this focus and that bronchus, and also on the number of other such foci which may be present, depend the

number of bacilli that are to be found in the sputum from time to time. One such small focus may result in the sputum simply swarming with T.B.; or it may be that a large focus not in free and direct communication with a bronchus results in the presence of very few or may be no T.B. at all in the sputum.

Sometimes especially where the lesion is deep seated and in free communication with a bronchus the finding of T.B. in the sputum may be the first evidence of the disease and which may thus show itself long before any other definite physical signs appear. (V Case 26). The moral to be drawn from this is, that in all doubtful cases and even those which if so must apparently be in the very earliest stage, to examine the sputum carefully if it can be got, and if it cannot be obtained at once, to use every effort to obtain a specimen as soon as possible.

T.B. are earliest and quickest found in the purulent looking nodules which are usually present in the sputum of a phthisical patient, and of these especially in those stained with blood if they should happen to be present.

Although the finding of T.B. in the sputum does not indicate the stage of the disease, it means that the disease as said before is active. I suppose that this must be accepted as a definite rule, yet I am not convinced however that like many other rules there may not be an exception to it. I have seen a

case who was supposed to have had T.B. in the sputum for years and yet when all the facts that were to be ascertained were taken into consideration hardly any other conclusion than that the disease was quiescent could be come to; the only point against this conclusion was this very fact of the sputum containing T.B. (V. Case 25). The human body as is well known can act as "host" to the Diphtheria Bacillus, the Typhoid Bacillus, the M. Meningitis and probably others, without any apparent ill effects showing themselves; these are the so-called "Carriers". It is not beyond the bounds of possibility that we may also have Tubercle Carriers of a kind; if so, I must admit that such cannot be common yet one cannot as yet go so far as to totally deny the possibility.

A most important point in estimating the degree of activity of the disease is the accompaniment or otherwise to the T.B. of elastic fibres. If both are present then the inference is that the disease is active and advancing; the quality of elastic fibres in the field gives some idea as to the rapidity of the destructive process.

It is not the intention of this thesis to go into the question of the differences between the varieties of the T.B., human bovine and so on; whatever the type found, for diagnostic purposes the fact of the finding alone is sufficient. I am not going to describe in detail the Ziehl-Neelsen method of staining by carbol-fuchsin depending on the acid-test of

properties of the bacillus; every medical man knows this and if not he should. This method of course should always be tried first and not only once tried, but frequently tried with many films and if possible with more than one specimen of sputum. If this fails the next step is to proceed to the use of one or more of the so-called "concentration" methods.

Probably the first to employ on concentration method was Biedast^① in 1886 who attempted to dissolve the sputum by the use of caustic soda and gentle heat. Sedimentation was allowed to take place after this and then films made from the sediment and stained. Biedast showed that organisms other than the T.B. were in this way got rid of.

It was not until 1908 however that the matter received serious consideration and that more successful methods came to be employed, notably the method of Uhlenhuth^② who introduced the "Antiformin" method. Antiformin consists of a mixture of sodium hypochloride and caustic soda which has been used in the ~~vats~~ by brewers for a long time for the purpose of cleaning the copper vats and utensils in connection with their trade. It has a powerful solvent action on all organic material like fat, dry yeast and the scum that forms on the top of the beer when in the vats and which is apt to soil the vessels. When antiformin is added to the viscid ~~te~~^{tr}acious sputum rapid solution soon occurs with the evolution of gas bubbles which escape from the mixture. The resulting liquid is of a

yellow colour containing a certain amount of mucus like sediment. The result of this process in the contained organisms is that all save the T.B. are destroyed, this escaping by reason of the fatty capsule which as is well known surrounds it, and which has been so well demonstrated by Bulloch and MacLeod⁽³⁾ who succeeded in actually extracting^a chitinous wax from cultures of T.B. by the use of hot alcohol and ether. It has been demonstrated that it is possible for the T.B. to live for days without losing any of its virulence. Modifications of Uhlenhuth's method have been adopted chiefly with a view to hastening the sedimentation process, the main principle for this being the addition of strong alcohol in bulk and centrifugalising the mixture. Personally I have adopted the modifying method of Lorenz⁽⁴⁾ which consists in bringing the homogeneous mixture up to the boiling point, centrifuging for about ten minutes and then examining the residue. The details are as follows:-

1. Add 30 cc of Antiformin Solution (15%) to 5cc of Sputum.
2. Shake vigorously from time to time for about one hour until the mixture is homogeneous.
3. Bring up to the boiling point.
4. Centrifuge until sedimentation is complete.
5. Pipette off supernatant liquid and make films from the residue.

I have often been able to find T.B. by this method when I could not by the ordinary Ziehl-Neelsen.

(V. case 38). Seeman's⁽⁵⁾ idea of using a film of the

original sputum as a fixative is good; it allows of other organisms which may be present also being demonstrated.

Ellerman and Erlandsen⁽⁶⁾ claim even better results from their method than Uhlenhuth's. They use a 6 % solution of sodium carbonate as a diluent. The process is as follows:-

1. To 5 cc sputum add 25 cc of the diluent.
2. Incubate for 24 hours.
3. Decant and centrifuge. Repeat this process once again.
4. To residue add an equal part of a .25 solution caustic Soda.
5. Gently warm and shake up.
6. Centrifuge and examine the residue.

The obvious disadvantage of this method is the length of time required. I cannot claim any personal experience of this method.

In spite of these concentration methods it is well known that it is often impossible to detect T.B. in what are undoubtedly tubercular lesions, as proved by the fact that when material from such lesions are injected into a susceptible animal it is shown to be highly virulent. The reason that used to be advanced for this was that probably the T.B. were extremely scanty or that they existed only in the form of spores. The recent researches of Much⁽⁷⁾ however have thrown quite a new light altogether on this point.

About four years ago when I began to take up the

study of Tuberculosis more specially, I wrote to Dr. Carnegie Dickson asking if he would be so kind to give me any suggestions with regard to a special branch of the subject which would lend itself suitable for a thesis. In his reply he strongly urged my following up either these recent researches of Much or the Wassermann Principle of Complement Fixation as applied to Tuberculosis.

With regard to the former most of my material for investigation was the sputa of phthisical patients which having proved Negative to the ordinary Ziehl-Neelsen staining;⁹ proceeded to examine by the Gram-Much method as described in Muir and Ritchie's Manual of Bacteriology (5th Edition. P 265.). My first conclusion was that the method was of great value and I am afraid that I got many positive results which in the light of later experience were not justified, because I had made the mistake in taking for Much granules small cocci which are of course often present in a sputum. At this time I was not practically acquainted with the Antiformin method; had I made it the rule to adopt this before proceeding to staining by Gram-Much the possibility of this error would have been eliminated. I do not find the granules so often now as I thought I did when first looking for them, nevertheless I consider the Gram-Much staining method of great value because I have undoubtedly been able to demonstrate these granules at times in the sputa

of patients who are beyond question phthisical and yet T.B. could not be found by the ordinary Ziehl-Neelsen. My own personal experience does not allow of my going beyond this bare conclusion because of the limited number of examinations that I have as yet been able to carry out. From what I can gather from others however I think that before giving up the microscopical search for T.B. most Bacteriologists agree that the Gram-Much method should be tried after previously subjecting the sputum to the antiformin process.

About three years ago I had the pleasure of listening to an address by Prof. Sims Woodhead to the Sunderland Division of the B.M.A. on the "Control of Tuberculosis". In a subsequent conversation it was he who pointed out to me the possibility of my early mistakes in the carrying out of the Gram-Much method if the Antiformin or other such process was omitted. [With regard to the Wasserman Principle as applied to Tubercle, he informed me that they had already done a good deal of work on the subject at Cambridge and that the conclusion they had come to was that the test was of no practical value because no really definite information could be got from it as to what was or what was not an active lesion.]

Much showed that the T.B. can exist in at least three forms; first the ordinary as stained by Ziehl-Neelsen; secondly as a fine rod shaped granular organism which is not acid-fast; and thirdly as free

granules occurring in the field in irregular bunches, or as isolated particles which however are not acid-fast and are Gram positive. The exact value of these granules was proved by Much by injecting the material containing them into the peritoneal cavity of a guinea-pig, and found that after three days sojourn there, ~~in~~ that they had recovered the acid-fast properties and that they later gave ^{rise} ~~rise~~ to acute tuberculosis. It can hardly yet be established that these experiments and conclusions have withstood all criticism; for example it must be very difficult indeed to say definitely whether there might have been a few acid-fast bacilli in the original material or not. It seems possible to conclude definitely however that there is a form of T.B. which is not acid-fast, as it has also been shown by Wirths ⁽³⁾ and others that it is possible for acid-fast T.B. on being injected into the peritoneal cavity of a guinea pig to lose their acid-fast character and so their powers of retaining the carbolfuchsin stain under the Ziehl-Neelsen method. This change was noted to have occurred more often in those bacilli which had been subjected to the action of phagocytosis. It is reasonable to assume therefore that the presence of such Gran-positive but not acid-fast bacilli in the material of which tubercular abscesses is composed, might be due to the action of autogenous ferments which have been generated in the abscess cavities; these ferments may have reached by digesting off the normal fatty capsule surrounding

the ordinary acid-fast bacillus.

Much showed three methods of modifying the Gram-Method, that known as the second or Gram-Much 2 however in the opinion of most authorities is held to give the best results. The technique is as follows:-

1. Stain in Methyl-Violet at boiling point for about three to five minutes.
2. Without washing apply Gram's Iodine for one to five minutes.
3. Drain off excess of Iodine solution and apply 5% nitric acid for one minute.
4. 3% H.C₂H₅ for ten seconds.
5. Complete decolourisation with a mixture of equal parts of acetone and alcohol.

The Gram-Much process proper is finished at this stage, but in practice I think it is better to counterstain the film with some other stain, as otherwise if no Gram-positive organisms are present, ^{just to show} the film is still present on the slide it is better to counterstain for a few seconds with a weak solution of carbol-fuchsin or saffranin.

⑨
Spengler's "Splitter Bodies" appear to be identical with Much's granules. Spengler demonstrated these "Sporoid" bodies as he called them in all the types of tubercle bacilli which he described viz. Typus Humanus-B~~ovis~~ (Koch); Typus Humanus-longus (Spengler); and Typus-Bovinus. Spengler's picric acid method is said to stain T.B. even when the Ziehl-Neelsen method fails to reveal them, and it also stains the "Splitter". The stain used in the method is Carbol-Fuchsin, and

this is fixed in the bacilli by means of picric acid; 15% Nitric acid is used as a decolourising agent. As yet I can have no personal experience in the use of this method. These granules according to Spengler indicate the last stage of the destruction of the bacilli and probably indicate a high degree of resistance on the part of the injected individual.

The enormous importance of sputum examination as a means of diagnosis is thus clearly evident, always bearing in mind however that whilst a positive result clinches the diagnosis, a negative result if it can be called a result means nothing.

If sputum from any suspected case is available for examination it must be most carefully examined in the manner above described. If the result after patient search is negative, another specimen should be tried and hence it is best whenever possible to make a daily search of the sputum for T.B. as long as there is any doubt; it often happens that the T.B. may be found after repeated attempts, and in this way the diagnosis is definitely settled. Even sputum which is thin and clear should not be discarded when no other is available, as it is sometimes possible to find T.B. even in such specimens if carefully and diligently looked for.

If no sputum can be obtained in the ordinary way then Holt's ⁽¹⁰⁾ method may be tried; this is valuable sometimes in the case of children who often swallow their expectoration. A swab of wool or muslin held

in forceps is used to tie^R the pharynx, in this way a spasmodic cough is caused, at the same time the expectoration resulting from which, is caught up in the swab which is then examined. On one occasion in the case of a young child when every other method failed I was able to pick out thick masses of sputum containing T.B. from the vomit which had been obtained by means of a dose of hot Ipecac. Wine.

Finally when all these means have failed of demonstrating the presence of T.B. in the sputum it may sometimes be justifiable to resort to animal inoculation; the sputum may then only be shown to be virulent by the effects produced in the animal. Changes may show themselves at the seat of inoculation as early as one week after injection, but on the other hand it may require even as long as eight weeks to definitely establish that the animal is suffering from tuberculosis. Usually however if the inoculated material is virulent definite evidence of the fact can be obtained if the animal is killed and examined at the end of the third week. It may then be possible to find T.B. in the nearest lymphatic glands draining the injected region; if the peritoneal cavity be the region inoculated then the inguinal glands are usually the first to reveal the presence of the T.B. Elch⁽¹¹⁾ improved upon this method still further by showing that if at the time of injection the inguinal glands of the animal were crushed between the fingers and thumb, it was possible to demonstrate T.B. in these glands as early as nine days after. The antiformin method is here of great value in dissolving out gland

substance when searching for T.B. Such a method is of great value which thus renders it possible to clearly establish a definite diagnosis by animal inoculation within so short a time; formerly it took almost as many weeks as it now takes days before such a report could be obtained from the laboratory.

THE ALBUMIN REACTION OF THE SPUTUM.

The presence of albumin in the sputum is of considerable diagnostic value. In the first place when dealing with a large number of sputum specimens a sharp division can at once be made into two classes i.e. those that contain albumin and those that do not.

Sputa with no albumin as a rule consist of a more or less abundant secretion from the bronchial mucous membrane and correspond clinically to acute or chronic bronchitis or emphysema. The presence of a sputum in albumin however indicate the existence of a deeper process inflammatory and exudative rather than a superficial catarrh and so probably allows of the exclusion of simple bronchitis altogether as the cause of the condition.

Albumin is constant and often very well marked in the sputum of those suffering from pulmonary tuberculosis; it also occurs however from time to time in patients who are suffering from chronic heart affections and chronic Bright's disease; it is also present in cases of Acute Lobar and Acute Broncho-pneu-

monia apart from any possible tubercular cause of the conditions. These latter diseases probably only give rise to the condition occasionally and so is temporary or periodic in character. Sputum of a constantly albuminous nature is strongly indicative of phthisis pulmonalis. The conclusions of Ridge and Treadgold⁽¹²⁾ after the analysis of over 2000 specimens are striking, they are as follows:-

1. All cases of Phthisis Pulmonalis practically always contain albumin in the sputum.
2. In those cases which are not associated with T.B. in the sputum, a negative examination for albumin on three consecutive occasions is strong presumptive evidence against pulmonary tuberculosis as the cause of the sputum.
3. The presence of albumin in doubtful early cases of phthisis helps to support this diagnosis.
4. The test is of considerable value in cases of chronic tuberculosis and emphysema as evidence of active disease.

There is no doubt then that the constant presence of albumin in a sputum is of high diagnostic significance, and recognising that the other causes of the condition as previously mentioned ought to be easily eliminated on clinical grounds, we have thus an easy method of arriving at a positive diagnosis. Unfortunately however the old proverb still holds good here, namely that you must catch your hare before you can cook it, and there are many cases of early active tubercle not associated with sputum at all or if so it is impossible to get a sufficient quantity for analysis. Rivière⁽¹³⁾ in his criticism ~~in~~ of the work of Ridge and Treadgold states that he is inclined to lay more stress on the

value of a positive and less on a negative result; ~~in~~ this may well be because of the logical principle that a negative ~~never~~ can prove anything definite and also because as said before the causes of an albuminous sputum are definite and few, and as also said before those other than tubercle ought to be easily eliminated clinically. From a practical point of view however I think a negative result is of great value; my reason for this is that one often meets with cases of acute Bronchitis or Bronchial Catarrh in what I may term phthisical looking subjects who however are able to keep at work even when attacked with these so-called 'colds'; the sputum from these cases is often abundant and râles and rhonchi can often be heard in the apical regions. One has often real doubts as to whether active tubercle may or may not be associated with such cases. My contention is that if these conclusions of Ridge and Treadgold are to be depended on, which up to the present I think they are, then the constant absence of albumin from the sputum of these cases is of great diagnostic value; the **inference** being that such a case is one of simple Catarrh only. The technique is simple and indeed is of little more trouble than the examination of urine. One or two precautions are necessary. The sputum should be collected into a clean wide necked bottle. The first sputum on awakening in the morning should be chosen, and it is important here that this should be expelled by coughing only, and that any nasal secretion sniffed into

the pharynx to be expelled by the mouth, excluded, as should also any sputum containing blood. The specimen should be examined fresh as soon as possible after collection. It should be poured out into a wide flat bottomed glass dish standing on a dark background and all clear mucus removed from it as far as possible. Rather more than twice the quantity of normal saline is now added and the whole acidulated with a few drops of acetic acid to precipitate the mucin. The whole is now stirred up with a glass rod and filtered. Albumin if present appears in the filtrate and is demonstrated in the usual way by boiling the uppermost layer in a test tube.

Since reading Ridge and Treadgold's paper in the Lancet two years ago I have occasionally tested the sputum for the reaction when there has been an element of doubt in my mind and in one or two cases I must say that I have derived considerable help from the method. The aid was more in helping to definitely decide what was not tubercle and what was, because personally I think that those cases associated with the active disease and an appreciable amount of sputum rarely present any great difficulty in their diagnosis for an albumin test to be resorted to as a special aid. It is for such reasons then that I disagree with Riviere and hold that, at least as far as the general practitioner is concerned, a constant negative result is clinically of more value. Illustrative of this the following case is quoted of a patient who often comes

to consult me:-

N.S. Age 53 years. Bricklayer. For a considerable time has been subject to a cough and shortness of breath; occasionally has more severe attacks which necessitate his lying up for a day or two, otherwise he manages his work fairly well. He expectorates a large amount of clear frothy sputum which has never contained blood however and no T.B. were ever found. Physically he is rather decrepit and broken down looking. He is thin weighing 9 stones 3 pounds for a height of 5 feet $9\frac{1}{2}$ inches. Chest measurements 32 inches to $33\frac{1}{2}$ inches. On percussion there is very slight blurring of ~~K~~önigs area on the left side but otherwise there is no definitely dull area to be made out. Breath sounds harsh with slight prolongation of expiration almost all over the chest; moist and dry accompaniments also generally present. The appetite is poor and there is a trace of albumin in the urine at times. The right border of the heart is out to the extent of about $\frac{1}{4}$ inch. There is marked retraction in the interspaces and the appearance of the chest is not emphysematous. When this man first came under my notice I was strongly inclined to think that I had a case of phthisis pulmonalis to deal with until I got a more complete history from his wife. She told me that he had been no different for the past four or five years and that attacks that laid him up for a day or two came on after drinking bouts to which

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he was peculiarly liable on pay days. I have tested the sputum of this patient for albumin on five separate occasions extending over a period of ten months, on no occasion was there the slightest trace of albumin. This patient probably belongs to the great legion of Chronic Bronchitis who practically live on nothing else but beer for food. I quote the case simply to illustrate the fact that if there was any doubt about its being tubercular in nature, the constant absence of albumin from the sputum would be a strong point against this.

ELASTIC FIBRES.

The presence of these in a sputum as said before is pathognomonic of lung destruction, and if present along with T.B. is indicative of advancing pulmonary tuberculosis. After examining for T.B. the sputum should always be examined for the presence of these fibres; indeed if desired the examination for both can be made at the same time as follows:- Add to the sputum an equal quantity of 10 % caustic soda and bring it up to the boiling point. Pour the swollen gelatinous mass into a conical specimen glass containing a little water and allow to settle; if necessary this stage can be expedited by the use of the centrifuge. Place a little of the deposit on a slide and examine under the low-power when elastic fibres are immediately recognised if present; they are sharply defined double-contoured wavy threads either single or

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branched; occasionally they may be in the form of a mesh work corresponding to an air vesicle of the lung. T.B. may be found on making films from the deposit ~~whi~~ which are stained and examined in the usual way.

CYTOLOGY OF THE SPUTUM.

As in the case of an examination of a pleural effusion, the principle is a differential count of the variety of cells to be found. A large proportion of lymphocytes is indicative of tuberculosis; the large mononuclear cells have their origin probably in the pulmonary alveoli. Ridge and Treadgold also made some interesting observations with regard to this subject. They regard that these mononuclear cells bear a close relationship to, if indeed they are not responsible for, the presence of albumin in the sputum. They have subdivided the cells into three classes according as to whether they are large, medium or small. The presence of nuclear degeneration in the cells is strongly indicative of phthisis. They describe a "dusty" appearance of the network of the nuclea which they regard as characteristic of phthisis and bronchiectasis. This appearance they claim bears some relationship to the stage of the disease being much more evident when destruction of lung tissue is marked. The technique recommended is as follows. The film is fixed by immersion in 1% chromic acid for two seconds and then washed. Next stain for two or three minutes in undiluted polychrome Methylene blue (Unna). Differentiate with 90% alcohol, wash dry and mount.

When stained in this way mucus becomes violet-red to artificial light whilst the sero-albuminous exudate appears as large violet drops.

These results of Ridge and Treadgold have not been confirmed to any great extent, but as their work seems founded on true scientific lines, it is probably that much valuable information on this foundation may be expected to accrue from it.

EXAMINATION OF THE BLOOD.

Examination for T.B.

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Prof. Lydia Radinowitchsch has published some details with regard to examination of the blood direct for T.B. She has produced evidence to show that tuberculin can liberate from a lesion a sufficient number of bacilli to render their recognition in the blood possible. Whether this work will lead to the establishing of a diagnostic method of any clinical value or not it is as yet impossible to say.

The Complement Fixation Test.

The Wasserman Reaction is the term generally used for this method of diagnosis. Although the great value of the principle has been chiefly seen in the diagnosis of Syphilis when a positive reaction is certain evidence of the syphilitic taint, it has also been used with the object of diagnosing other diseases which are also due to specific causes e.g. Tubercle, Typhoid, Glanders, Echinococcus Disease. The basis

of the principle rests on the fact that when an animal has injected into its blood-stream certain proteid elements, it reacts to them in a way calculated to destroy these foreign bodies resulting in the development on the part of the animal in question of what are known as 'Antibodies' which are present in the blood of the animal. If these antibodies can be shown to be present in the blood of any animal then proof exists of infection being present within that animal. For example an animal is injected with tubercle at once an attempt is made by the animal to form within itself anti-tubercle bodies to show that the animal is or has been injected with tubercle.

The material which is inoculated to produce antibodies is known as the "Antigen", and in the case of Tubercle or Syphilis this antigen and its antibody will only react on one another in the presence of a third substance called "Complement". Now Complement is a substance which is present in the circulating blood of every animal, whereas antibodies are only present in the blood of those animals which have reacted to the them i.e. an antibody is specific for its own antigen only. Complement is rapidly destroyed when the blood is heated to 56°C ; it cannot be isolated, yet it can be used in the medium of an animal serum, usually that of a newly killed guinea-pig. The serum of a tubercular animal having been mixed with tuberculin is now to be tested for its capacity of absorbing complement which as already seen it can only do in virtue of its possessing a requisite amount of these

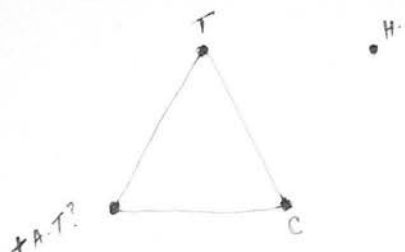
immune or antibodies. If now this can be demonstrated ~~in~~ in vitro i.e. that combination takes place when three bodies are brought into contact with one another, then knowing the actual presence of two of them the presence of the third is detected with certainty, this in the case of tubercle would be anti-tuberculin which in its turn indicates an infection of a tubercular nature being or having been present in the suspected animal. Thus apparently it becomes possible in such a manner to diagnose the presence or absence of infection with mathematical precision.

In the cases of tubercle and syphilis however ~~in~~ there is no direct means of showing that this triangular combination has taken place in vitro; use is made therefore of another group of antibodies which also react in the same way, but with the difference that visible changes occur when antigen, antibody and complement combine. The antibodies used for this purpose are known as "Haemolysins". A Haemolysin present in the serum will combine in the presence of complement with the specific corpuscles which have given rise to its formation; the result of this is that these corpuscles become broken up or "laked" whilst at the same time the whole mixture becomes red and transparent. If complement is absent this reaction cannot occur for the reason stated that antigen and antibody can only combine in the presence of sufficient complement i.e. the combination is essentially a triangular one. With the presence then of corpuscles and their

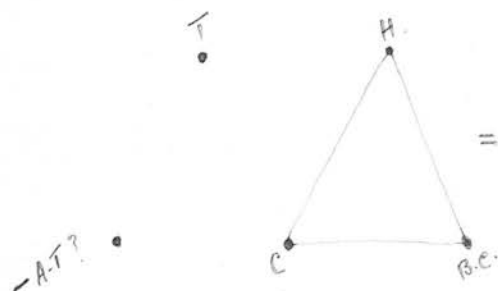
specific antibodies also present in a mixture, it becomes possible to visibly test for the presence or absence of complement indicated respectively by the occurrence of haemolysis or sedimentation of the corpuscles. If then a certain amount of complement is present along with anti-tuberculin and if enough antigen or tuberculin is added to ^{these} a triangular combination will occur resulting in the using up of the complement, so that if haemolysis and corpuscles are subsequently added no combination ^{between} these latter two can possibly take place because there is no complement to bind them. The nature of this state of matters would be indicated by the fact that no laking or haemolysis of the corpuscles occurs; this is a Positive Reaction i.e. antituberculin was present in sufficiently strong amount in the serum of the suspected animal i.e. the animal was tubercular.

If now the suspected animal is not tubercular, then no anti-tubercle bodies are present, no triangular combination between these, antigen and complement occurs, so that complement is free in the mixture. If haemolysis and corpuscles are now added these quickly combine in the presence of the free complement, this is indicated by the laking or haemolysis of the corpuscles the essential of a negative reaction i.e. the suspected animal's blood contained no tubercle antibodies and was therefore free from tubercle infection. The positive and negative reactions are graphically represented in the following diagrams:-

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= no Haemolysis or
Positive Reaction



= Haemolysis or
Negative Reaction

In theory this argumental proof seems almost infallible but at first it was unfortunately a fact that antituberculin could not be found in the blood of many undoubtedly tubercular persons; it could be demonstrated however in the serum of those who had undergone a course of tuberculin treatment. More lately however with improved technique antituberculin has been found present not only in the blood of those suffering from phthisis but also in the blood of persons who have exhibited not the slightest clinical sign of their being tubercular, although technically such cases must be considered as being tubercular. All that this goes to prove then is the almost total universality^{re} of tubercle infection amongst civilised people, and an effort on the part of those of us who have escaped clinical signs of the infection to resist such infection

by the development within us of these anti-tubercle bodies. The finding of these antibodies then, in fairly large amount may warrant one in coming to a conclusion, which at first sight seems entirely paradoxical, namely that such a person is free from infection and is also not likely to fall a victim to it, always understanding however that in this case one is speaking of the disease from a clinical point of view only. In such a case tubercle has acted like many of the acute infections, an attack of which has produced a more or less permanent subsequent immunity; although it differs from these in the fact that the initial infection has not been associated with any definite clinical signs. One can thus understand then that from many points of view an adult may have enormously benefitted because in his childhood he was threatened with certain manifestations of tuberculosis and was treated accordingly; it is quite ^{possible} that there are a great many of us who at present owe their immunity to tubercle infection to the fact that we were subjected to mild attacks of the infection as children and which were subsequently resisted. In those of us who have no history of any such indications in childhood or early youth, the case may be very little different because it cannot be denied that we have all been exposed on occasions to much the same amount of infection, but because of the suitability or perfection of our environment or inheritance, allowing of our

successfully resisting any outward signs of the infection which are commonly known as Tubercular Disease. In the former case resistance has made itself evident by clinical signs, in the latter the resistance has been associated with invisible phenomena; in both cases healthy manhood may be attained with the development of a large amount of anti-tubercle bodies in the blood serum. Incidentally I may mention, that to my mind, this hypothesis has a most important bearing in the production of an immunity by tuberculin; there is the school who treat their cases of phthisis by what is comparatively speaking large doses of tuberculin to which reactive symptoms occur; there is another school to which I myself belong, who treat by small doses given over a long period, without the production of reactive phenomena. If, as I have said before, we as healthy adults owe our immunity, as I think we do, to our successfully and invisibly resisting the omnipresent infection during childhood and adolescence then the latter school have certainly, to my mind, more rational grounds for their line of procedure.

When the principle of complement deviation is considered in its relation to the diagnosis of Syphilis then the value of the reaction is much more important, because in this case all we want to know is that the patient possesses in his blood serum such syphilitic antibodies; the question whether syphilis is active or latent does not come in for consideration.

here at all, one is satisfied from a diagnostic point of view that the patient exhibits a positive or negative reaction as the case may be. For this reason then I hold that Emeritus Prof. Chiene is, or was wrong when he used to teach us that the reason why many escaped Syphilis was because of an inherited immunity i.e. that the race was becoming syphilised. The study and careful consideration of the Wasserman Reaction may warrant the conclusion that the civilised peoples are becoming Tuberculised but certainly not Syphilised and this is the reason why the Wasserman Reaction is of enormous value in the diagnosis of doubtful Syphilis and of so little value as yet at any rate in the diagnosis of what may be doubtful clinical tubercle.

When is the border line crossed, when the immunising process breaks down and an infection becomes in reality a disease? This is the question which one would like to have answered definitely and as soon as possible in every case, and whilst the principle of complement fixation and deviation can undoubtedly reveal the presence of tubercle it cannot unfortunately settle what is active and what is latent, and which as so often said before is the so essential point to be determined in the diagnosis of all tubercular conditions.

The principles underlying the Wasserman Reaction and the great amount of work, really beautiful in its conception which has resulted from it, is in my opinion worthy of placing medicine at the head of the

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list of the true sciences; Like many of the principles in the true sciences the Wasserman Reaction has not as yet, as far as tuberculosis is concerned, found its practical application from a diagnostic and clinical point of view. One cannot say however that the present stage is the final one, for how often in medicine has many a valuable agent or principle e.g. Lister's or Koch's Tuberculin been condemned before at length its true place was recognised and granted.

The Opsonic Index in Diagnosis.

The capacity of the leucocytes for the phagocytosis of bacteria was first demonstrated by Leishman in cases of simple staphylococcus infections such as Acne or ⁽¹⁵⁾ Eils. He showed in such cases that the capacity of the leucocytes for ingesting staphylococci was much below that of the leucocytes taken from a normal healthy person. Leishman's experiment was a simple one and was performed as follows. Into a fine capillary pipette about half an inch of the sample of blood to be tested is aspirated; an air bubble is allowed to enter, and then next, about half an inch of a thin emulsion of the bacterium to be tested is aspirated. The two fluids are mixed by expelling them on to a slide and aspirating them into the tube again; this is done two or three times. Place a cover glass over the drop and then incubate for 15 minutes at 37°C. An exactly similar procedure is done with the blood of a healthy person. Film preparations are made of the two specimens stained and then compared under the

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microscope as regards the ingesting power of the leucocytes for the bacteria. The number of cocci in 50 or 100 leucocytes are counted in each case and the two so compared. In this way an estimation of the phagocytic capacity of the blood is arrived at.

According to Wright⁽¹⁶⁾ the phagocytosis that occurs in this manner outside the body is not a simple one and depends on the presence in the serum of substances which he called "Opsonins" (Gr. Opson-sance) and as the word indicates, these in some manner prepare the way for the ingestion of the bacteria by the leucocytes. For practical purposes it is quite immaterial whether they act by stimulating the leucocytes or by lessening the resistance of the bacteria. Wright and his workers studied the action of these opsonins in connection with the treatment of infections by vaccines. The technique for the estimation of the opsonic index depends on the preparation of the following:-

1. A bacterial emulsion.
2. An emulsion of living leucocytes.
3. The serum of a normal healthy person.
4. The serum of the supposed^f injected person.

It is not my intention here to go into the practical description of these preparations as this is given in every text-book on Bacteriology in full detail.

Two preparations are made using the same bacterial emulsion and the same leucocytes in both cases but with the two different sera mentioned above however. The phagocytic capacity of the serum from the supposed

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injected person compared with that of the healthy individual determines the "Opsonic Index" i.e. it is a ratio.

The variation of the Opsonic Index in cases of tuberculosis is extremely interesting. The special feature about it is that it is extremely variable especially in those cases where the disease is progressing rapidly, and also in those patients who are taking exercise. This variability is supposed to be due to Auto-Inoculation; bacilli or their toxins are set free from the lesions to get into the general circulation, where they act in exactly the same manner as an injected dose of tuberculin does.

Out of the vast amount of work that has been done with relation to the O.T. in cases of phthisis, three prominent facts seem to emerge. First the index of normal persons is always found to be within the limits of .8 & 1.2 (Bullock⁽¹⁴⁾). Secondly in phthisis pulmonalis the index is either below or above this normal limit, and is subject to marked fluctuations. Thirdly tuberculin has a marked effect on the index; there is first a fall -negative phase-, next a rise -positive phase-, and lastly a fall to the original level or very often to a level slightly higher than the original.

By the application of these points to any selected case then it ought to be possible to diagnose any case with accuracy. If, in the study of any case, a series of opsonic index estimations is made under varying

conditions the following questions submitted ought to be answered:-

1. Is the index constantly within the normal limits as stated above?
2. If not is it high low or fluctuating?
3. Can specific auto-inoculation be produced by exercise?

The answer to these questions definitely decides the absence or presence of tubercle; not only this but what is much more important it decides whether the tubercle is active or otherwise. With normal individuals the index remains within normal limits at every examination i.e. at different periods of time and under different conditions. In tuberculosis the index may be high or low, and if it is normal at one time it never remains so on repeated tests. Definite fluctuations due to spontaneous auto-inoculation indicate tuberculosis. Definite fluctuations as the result of exercise or exertion i.e. artificial auto-inoculation indicate tuberculosis; in this case all small fluctuations must be disregarded or at most only looked upon with suspicion.

It would seem therefore that in the determination of a series of opsonic indexes that we have an exact method of diagnosis in all doubtful cases. Unfortunately however, experimental differences are so very large in this class of work that individual observations are always liable to be exceedingly misleading e.g. to quote the classic criticism, if the same samples of blood from the same patient and under the same

conditions are sent to different pathologists for examination, widely different reports may result, and it is for this reason that the opsonic index method has so largely fallen out of fashion amongst general practitioners and others. The causes of these experimental differences are well worth some consideration because ~~even~~ if it must be admitted that with the best and most careful of workers a certain amount of experimental error is bound to occur, yet I do not think that this is enough to account for the wide differences that take place when the results of different undoubtedly skilled pathologists are compared. The researches of Morgan and Hayden⁽¹²⁾ when considered demonstrate to us that in the antigen i.e. the bacterial emulsion, there is at least one supreme factor which ~~would~~ alone would account for wide differences. These observers showed that the sensitiveness of the antigen may be varied at will from a low degree practically of little worth because of the narrow margin in which tuberculous sera tested by it are bound to vary, up to a very high degree whereby the tests became too sensitive so much so that, if depended upon, normally healthy individuals might become classed as tuberculous because of the small differences in their sera. This at once reveals a reason whereby the opsonic indexes estimated at two different laboratories from the same case might vary so much. It is exactly the same in the case of Widal reaction; ~~in~~ precise details as to the value of the culture of

Typhoid bacillus must be adhered to or else the agglutination test as far as the degree of dilution is concerned absolutely fails; no sane man however will deny the value of a Widal reaction in Typhoid. The preparation of the emulsion of the tubercle bacillus in the case of the O.T. may be a little more complex and as yet not universally standardised, but otherwise the analogy is exactly the same. If antigens of different standards are used by different workers then results are bound to differ and indeed it would be passing strange if they did not.

In spite of all this then, there are cases however, where the determination of the O.T. under different conditions is of great value especially when the phenomena of auto-inoculation which takes place after exercise is taken into account. Warren Crowe gives a series of 23 cases of phthisis where the O.T. was estimated with a view to diagnosis, and in every case he was able to definitely decide what would be otherwise a doubtful diagnosis. It is only right to mention however that other equally eminent workers have not obtained the same assistance from the use of the O.T. determinations. Personally I have never yet seen a doubtful case which has not declared itself by other means of diagnosis in a quicker time than it has taken to obtain a series of opsonic indices under the varying conditions required.

To sum up then it would seem that here again in certain cases valuable information may be gained from

the study of the O.T. As with all the other aids to diagnosis discussed so far however, the O.T. alone should not be relied upon. It is only when it is taken into consideration along with the clinical data that it best serves as a diagnostic ~~prop~~ and so becomes, in its true place a means of value and reliance. If divorced from the clinical evidence and relied upon as a means in itself there is bound to come a time when great difficulties will occur and mistakes arise.

THE AGGLUTINATION SERUM TEST.

Tests devised on the lines similar to Widal's Agglutination Test for Typhoid have been attempted with a view to diagnosing Tuberculosis in a similar way. The serum of tubercular patients has been found to exert an agglutinating action on the T.B. in suspension, giving evidence in so doing of the presence of immune bodies in the serum of the affected person. As in other agglutination tests however it is difficult to show any definite relationship between the degree of agglutinating power and the amount of immunisation possessed by the person from which the serum was taken. The method as a means of diagnosis is probably of very little value for the reason that it is often found that the serum of an active case is frequently devoid of agglutinating power, whilst on the other hand that of may be a long-standing quiescent or healed case often shows marked agglutinating power.

COBRA-VENOM ACTIVATION METHOD.

The test is carried out on much the same lines as the complement deviation method already discussed. Cobra-Venom alone in strong dilutions does not possess the power of haemolysing red blood corpuscles. Calmette however claimed that with the blood of 94 % of cases of tuberculosis, when added to the diluted cobra-venom, there resulted an immediate haemolysis of the red blood corpuscles. Other workers however have found the same thing happen with the blood of as many 50 to 60 % non-tuberculosis cases. This method therefore can have very little value attached to it.

PASSIVE ANAPHYLAXIS TEST.

Healthy guinea pigs are passively hypersensitised by the injection into them of blood serum taken from tuberculous patients so that when an injection of tuberculin is given to the animals the typical phenomena of anaphylaxis sets in. Although the reaction is generally positive it has been found that other sera often give similar results. The method is then devoid of any specific value.

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ARNETH'S METHOD OF BLOOD EXAMINATION.

This method is based upon variations which occur in the neutrophile white corpuscles of the blood in affected persons. Arneth divided the neutrophiles into five groups according as they contained one, two

three, four, five or more nuclei. In health he held that the percentage grouping was made up as follows:-

One nucleus	5 %
two nuclei.....	35 "
three "	41 "
four "	17 "
five & more.....	2 "

In the very earliest stage of phthisis pulmonalis, before the general health is appreciably affected there is no alteration in this form of grouping. As soon as general effects set in however, the ratio is altered considerably. Group 1 rises to about 15% Group 2 to about 46%. Group 3 falls slightly; whilst groups four and five fall more decidedly.

As the disease becomes advanced then Group 1 rises between 28 and 52%. Group 2 between 37 and 53%. Group 3 falls to 10%. Groups 4 & 5 disappear altogether.

Arneth claims that this method gives a good idea of the intensity of the infection and the patient's power of reaction to this. In favourable cases i.e. in those reacting well to treatment and are beginning well to recover the grouping of the leucocytes tends to revert to the same type as in health. The technique is simple, films of the blood are made and stained in the usual way by Jenner or Leishman. Two or three ^{hundred} neutrophils are examined and grouped according to their nuclei. This method would appear then to be of more value as a prognostic agent than a diagnostic, because as the author states no changes are observable in the early cases. One cannot assume that

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these nuclear changes are only characteristic of a tubercular infection, it remains to be seen how the grouping of the neutrophiles responds to other infections because after all it is probably that nuclear changes can only be reactive in character and that hardly specific to tubercle. I have as yet no personal experience of this method so must await the finding of others with regard to these points. I must say however that in a series of blood films taken from patients in the wards generally, that the grouping of the nuclei did not correspond to that given above for patients in health. I found no neutrophile containing five or more nuclei, in only one case was I satisfied that there was really four. The obvious criticism to this of course is that these films were not made from the blood of healthy patients; my reply however is that neither were they taken from clinically tubercular patients yet a departure from the so-called normal state occurs and unless the classification for comparison is something like definite for non-tubercular cases, one can hardly hope to gather much information as to definite changes in the case of the tuberculous.

EXAMINATION OF THE FAECES.

T.B. are often found in the faeces of those suffering from pulmonary tuberculosis. Swallowed sputum is probably the origin of the T.B. in this case, as

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they are much more often to be found in the stools of children than of the adult. The fact may be of diagnostic importance if it should be impossible to get a sample of sputum; this as said before however is very rare if care be taken. Even when found in the faeces it does not necessarily mean that the T.B. have come from the lungs, they may have originated from some lesion in the intestinal tract, so their presence at most only labels the patient as tuberculous. This is probably the reason why the examination of the faeces for T.B. is of so much more value in the case of dairy cattle than human beings. All that is wanted in the former case is the pronouncement that the cattle beast is definitely tuberculous. In the case of the human being we want to know much more than that; it is not very difficult as a rule to diagnose that a patient is probably tuberculous, it is much more difficult however to locate the exact seat and condition of the lesion, which after all is the only diagnosis which is of any practical value when the treatment of the patient is considered. When searching for T.B. in the faeces the antiformin method is of great value.

EXAMINATION OF THE URINE.

The more special point to be considered here is the Diazo-reaction of Erhlich. This again can hardly be said to have much bearing upon diagnosis. A

positive reaction is a bad sign in cases of phthisis and usually indicates rapidly advancing disease. For the reaction to disappear under treatment of the patient is supposed by some to indicate an improvement in the condition of the patient. I do not agree with this however as I have seen the reaction come and go in a patient who was rapidly going from bad to worse. There are many cases of phthisis again which proceed to a fatal termination and in which the reaction has never been found to be present at all.

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RADIOGRAPHY IN DIAGNOSIS.

"Every Mother's duckling is a swan" (old saying)

It is not the intention of this thesis to go into the technical details of skiagraphic examination, because in the first place I am not qualified to do so, & secondly were I so qualified there is no reason for assuming that I should not become like most of the radiographers I have met, & think that in this branch of the subject alone, we have the method of diagnosis.

It is my honest opinion that if I had been a radiographer the bulk of my writing would have been taken up with this aspect of the subject only, whilst probably the fate of the other branches would have been to be dismissed with a few words, all the time being firmly convinced that I had given fair consideration to the diagnosis of ~~Phthisis~~ ~~Pulmonalis~~ ~~Phthisis~~ Pulmonalis. Expert X-Ray workers apart, I know that many general practitioners have for the time being at any rate, come under the spell & fascination of this branch of the subject. A second opinion may be wanted concerning a particular case & at once the patient is advised to consult the radiographer & have a plate or photo taken of his chest.

Without being cynical, I think the conditions & lines of thought leading up to this standpoint is in the case of many an interesting psychological study.

Chest work in one form or other bulks largely in the practice of the general medical practitioner. Now chest work is difficult, thereby I mean more especially, that it is laborious & takes up a great deal of time. As a rule the successful general practitioner is a busy man; when he comes to deal with a heart case the stethoscope as a rule certainly & quickly reveals something definite; & a murmur is heard, it may mean much, it may possibly mean very little, but at any rate it allows of a definite diagnosis being given the patient or his friends. If the patient gets worse no reflection can come on the medical practitioner because of the "I told you so" argument. When we come to deal with the lungs however, the case is very much different; here the stethoscope in an early case gives very little help, or if it does the aid given is not in the form of a loud ostentatious murmur which cannot be missed; again, if a murmur is detected its significance cannot be described to one disease only. Percussion, I am afraid, has become a lost art with many practitioners, who thus through force of circumstances are landed "upon the fence" Clinical intuition, Clinical insight, clinical diplomacy, call it what you like, may allow of the practitioner getting onto the right ^{side}, in many cases, but often ~~so~~ at the risk of being called an alarmist or pessimist; even this however may fail. To sum up then such a Medical man, & I have seen many, if honest with himself, is in many cases not easy in his own mind with regard to a particular patient.

Sending the latter to a consultant may overcome the difficulty, but often it does not, because one cannot always be sending such patients to a consultant, whilst even he may not always be able to give a definite diagnosis in a doubtful case on examining such for the first time. Now the radiographer comes on the scene; he claims that a plate or photo will reveal the exact condition of that patients chest; at last there is something definite to go upon; one can see the lesion or lesions, surely there can be no mistake; mournful to relate the patient has to be told that his lungs are affected, a definite diagnosis can be given.

There is another question however, which I find is not so easy to get the X-Rays man to answer. Can he assure me that a certain patient has not got phthisis? In other words I want a definite report that the patient is not clinically tuberculous.

The radiographer will quite confidentially state that the lungs of so & so are affected, but in the case of a fair number of doubtful cases examined by the rays I have never yet been assured that any one of them was absolutely free from clinical tubercle, when the after history of the case or cases subsequently proved that this was most likely to be the case (see Case XXX11 & photo)

This is the old story again, tubercle can be diagnosed definitely be this means, so it can, & it can also be diagnosed definitely be other simpler means. The diagnosis of early active tubercle - clinical tubercle - cannot be definitely

diagnosed by the use of the X - Rays alone. The clinical picture again plays a much more important part & radiography divorced from this evidence is useless, it is worse than useless, it is positively misleading. Now I do not wish it to be thought that I am hostile to the use of X-Rays, because this is not the case. I have often received valuable information from their aid, both from a diagnostic & prognostic point of view. I think highly of this means as a special aid when taken into consideration with the rest of the clinical evidence; as will be seen later, when considering the diagnosis of phthisis pulmonatis in children I think that most valuable information & assistance is to be derived from their judicious use. Speaking generally I think that the clinical evidence which the rays especially support, is that which is derived from percussion. It is my invariable rule to have the patient screened in the case of these early afebrile cases which have not definitely declared themselves by other means & where percussion taken into consideration, with may be loss of weight, anaemia, slight cough, or may be lack of tone, warrant one in having at least a strong suspicion of commencing active mischief; this I consider to be the line of procedure to be adopted in the examination of certainly one class of tubercular lung disease in children.

Apart from the latter class of cases however what radiography amounts to in most of the cases is that it becomes a substitute for percussion, or in other words when in doubt about one's own percussion powers

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have an X-Rays plate taken. In my opinion this is the reason why today radiography is so popular amongst so many general practitioners. Like the too frequent use of tuberculin for diagnostic purposes, it is an admission conscious or otherwise of one's lack of clinical powers, at any rate as far as the chest is concerned. I do not mean to say thereby that X-Rays are not necessary, but I do mean to say & maintain that for diagnostic purposes there is certainly no foundation on fact for the excessive popularity which they hold in some quarters today.

I lay emphasis on the words "for diagnostic purposes" because in the realms of pathology radiography has played a most important part in lung conditions.

Von Behring⁽¹⁾ was once looked upon as a false prophet when he maintained that in many cases adult phthisis was only the final stage of a childhood infection; the skiagram has gone a long way towards proving the correctness of this view.

Again, when the progress is under consideration, I have occasionally seen cases where the physical signs especially those of auscultation, were so marked that one would have concluded that the disease was well advanced, yet when a skiagram has been taken it has proved that the lesion was mostly a surface one & not at all advanced at that, (see photo). Again I have seen cases where the physical signs have been remarkably scanty, yet the rays have shown the existance of advanced disease centrally situated, such a condition of necessity being of grave prognosis (see photo)

Whilst these facts have no direct bearing upon the actual diagnosis of phthisis pulmonalis, I mention them if for no other reason than to show that I am not hostile to the proper use of X-Rays, as one might be apt to imagine from some of my previous remarks.

Although the clinician cannot be expected to be expert in the technique of radiography, I am of the opinion that he should have a fair working idea at any rate of interpreting a plate or photo, & also of the appearance on the ~~ph~~luorescent screen. At first, I had to accept the radiographers' view on everything connected with the screen or plate, for the simple reason that I knew nothing at all about the subject. I quickly came to the conclusion that in many cases there were suspicions for thinking, not that I was getting too little information, but that I was getting too much, & that I had better get an idea of the subject myself.

Before we can recognise abnormal appearances, the appearance of the normal chest must be carefully studied. The Hilus Shadow must be carefully noted, as also the normal shadows caused by the large vessels & bronchi. Shadows from calcareous glands may often be seen, but these do not necessarily mean very much. Shadows are normally present in the apical regions & over the outer borders of the lung caused by the normal presence of thick layers of muscle & by the internal border of the scapula, the two sides may not be exactly alike. The shadows may be naturally deeper on the right side. Arborisation of the tubes &

vessels may be followed even up to the lung surface. Fat obstructs the rays to a certain extent, so do also the mammae in the case of women. The diaphragmatic shadow is normally higher on the right side.

All these points must be taken into consideration, & also such points as temporary difference between expansion of the apices on the two sides. There are also differences depending on the special technique of different operators, the position of the plate, the length of the exposure & lastly the development of the plate. Regarding these latter points I do not profess to much personal knowledge, as holding such more within the province of the expert.

When a case of phthisis is up for examination the following considerations must be carefully born in mind when interpreting a plate or photo:-

1. The healthy lung substance offers practically no resistance to the rays. The appearance therefore is dark ribs & fairly clear interspaces.
2. Excess of air in the lung results in an abnormal clearness of the interspaces.
3. Diminution of air causes relative consolidation of the lung & therefore a certain degree of obstruction to the rays so resulting in a shadow of varying depth on the screen or skiagraph.
4. variations in the amount of air within the lungs affects the position of the heart, & also the overlying ribs. Thus the width of the intercostal spaces may be abnormally wide or narrow.

5. Fluid in the pleural sac causes a shadow varying in intensity according to the density of the fluid. With a clear apprehension of these facts, & also bearing in mind the radiographic appearance of the normal chest as stated above, the clinician is enabled to deduce valuable evidence from a plate, or at any rate he is in a position to appreciate the value of the expert radiographer's report which accompanies the plate or photo of any particular case under consideration. For example, supposing a case of early involvement of one of the apices is under consideration, the screen appearance usually reveals marked limitation of the diaphragmatic movement on the affected side. In addition to this it will probably be found that the affected apex is rather less translucent than its fellow of the opposite side; this is specially noticeable on deep inspiration. In the case of old standing lesions with accompanying fibrotic changes there is practically no illumination, a deep shadow showing itself corresponding to the seat of the lesion; in addition to this there may be the roof-filing arrangement of the ribs, caused by their approximation to one another over the seat of the lesion. Gradations of varying amount may occur between these extremes, depending on the condition & activity of the lesion.

Whilst these facts are most interesting indeed, I am not going to argue that all this information is new; the careful, & observant clinician was in all likelihood aware of the conditions causing these appearances long before the X-Rays examination took place.

All the same however, such evidence is markedly corroborative of that obtained clinically from the results of careful inspection palpation & percussion. It is as if a telescope had been brought to bear upon the scene & so allowing of a much clearer view of the underlying conditions.

From a diagnostic point of view however, it can hardly be held that in the majority of cases such evidence has done any more than make the diagnosis more certain. It is possible that in a few cases appreciable help might be given in settling an otherwise doubtful diagnosis. this is more especially likely to happen with regard to a certain class of cases which are met with amongst children, & which will be discussed later on. My main contention however is, that the more the clinician can educate & trust to his own powers of observation, the less often will a radiographic examination be necessary for diagnosis. Given a man, who, for one reason or another is unable to appreciate symptoms and to apply the special senses with which nature has endowed him, then the best thing he could do would be to install an X-Ray apparatus and skiagraph every chest case which came under his care, even then a great danger would likely lie in the probable diagnosing of many patients as suffering from phthisis pulmonatis who were not. I hereby assume once again that the term "phthisis pulmonatis" means active or clinical Tuberculosis of the lungs.

In conclusion then one must hold that it is quite

impossible for radiography to replace ordinary clinical examination. It is more than probable that the X-Rays alone are quite unable to differentiate between an active or latent lesion. They may confirm the diagnosis in a doubtful case, they may give great help in determining the extent of the disease & also in showing whether the lung of the opposite side is free or secondarily involved to any extent. This latter point is a most important one, as pathologically it has been that the seat of the commencing secondary lesion in the lung of the opposite side is usually situated deep within the organ. For these reasons it is to be expected then, that although a case has been apparently & justifiably diagnosed on clinical grounds alone, as one of early ~~border~~, yet the cautious man fearing & knowing how often the disease may be ^{more} advanced than the clinical signs would lead one to suppose, seeks, before giving a definite prognosis, the assistance of the X-Rays. It may then be found that although percussion & auscultation have failed to reveal a lesion in the lung of the opposite side to the commencing disease, yet the ~~rays~~ may at once detect this to be the case, a point which is of the utmost importance when considering the prognosis of any particular case.

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THE DIAGNOSIS IN CHILDREN.

"Principiis obsta. Sero medicina paratur cum malo per longas convaluere moras". (Ovid.)

Tuberculosis of the lungs in children until, comparatively speaking a short time ago, was always considered as a very rare disease.

I suppose a recently qualified man might regard this statement as rather surprising. The explanation probably lies in the fact that it is only comparatively a short time ago, since the special study of children's diseases was first seriously considered. Fifteen years ago there were very few indeed of the palatial establishments that one finds nowadays in connection with every large town, which have been erected for the housing and care of the sick child. It was not very long after the occurrence of such events before the medical officers in charge of these institutions, whose attention became naturally more specially directed toward the study of children's diseases, pointed out that the clinical picture of pulmonary tuberculosis in children was in many respects very different indeed to that observed in the case of the adult.

There is no doubt also that when studying the disease in children, one is at the same time

considering the "fons et origo" of many cases of the disease in adult life. There is still a good deal of difference of opinion regarding the extent to which the lungs are involved in children. The explanation of this probably lies in the methods of examination adopted; if clinical means only are adopted then a low percentage of cases are diagnosed, this rapidly mounts up when the more special aids to diagnosis already discussed are resorted to, such for example as Laboratory findings. Tuberculin-reactions and the X-Rays.

It now behoves me to state that it is not my intention here to consider in detail all the aspects of phthisis pulmonalis as it occurs in children, because when one considers that whole volumes have been written upon this branch of the subject alone, it thus becomes quite impossible, even if I were qualified to do so, to do more than touch on a few main points and especially those which are more concerned with the question of diagnosis. However as I have always a few childrens beds to take charge in hospital, it has always struck me that I should be doing less than my duty if I did not at any rate keep an attentive ear to the teachings and counsels of those, who both from experience and circumstances were infinitely better qualified

than myself to speak authoritatively on such an important subject. It has been well said that if you cannot be a "Johnson" try to be a "Boswell"; in other words cultivate the power of being able to appreciate and appraise the work of others; it is from a position like the latter only, that I can discuss such a subject as pulmonary tuberculosis in children. My reason for including the subject in this thesis is because I felt that such would be very incomplete without it.

Hamburger⁽¹⁾, and Mc.Neil⁽²⁾, Sheunan⁽³⁾, Mantoux⁽⁴⁾ have shown conclusively that during the first year of life tuberculosis rarely occurs, but after that it increases pro rata with the age of the child, until from puberty onwards somewhere about 80% of the population can show evidence of tuberculin infection past or present. The statistics of Von Pirquet reaction already given also go to prove this statement. It is most probable that in all these, the lung is the primary seat of the lesion.

A few years ago Greenwood⁽⁵⁾ published some interesting statistics with regard to the prevalence of pulmonary tuberculosis in children. Out of 338 school children examined 34 or 10% had phthisis pulmonalis. These figures at the time were looked upon with a great amount of dubiety

but in the light of modern knowledge especially that based upon tuberculin reactions and post mortem examinations they do not appear to be at all exaggerated, at any rate so far as an actual tubercular infection is concerned, whether these cases could all be called active disease or not I am not prepared to say. The development of the subject known as School Hygiene has at any rate gone a long way to show the prevalence of this disease amongst school children. Post-mortem findings have also largely assisted in proving that pulmonary tuberculosis is a fairly common disease in childhood.

Lapage and Mair⁶ found evidence of tuberculosis in 150 out of 323 necropsies and of these 14% showed pulmonary tubercle alone and 54% showed it associated with tubercular disease elsewhere. In 25 cases cavitation was present and in all cavity existed at the apex. These figures certainly ought to be proved that pulmonary tubercle is a common infection in childhood, yet when one comes to examine the death returns. As issued by the Register General we find that the deaths certified from Phthisis Pulmonalis become greatly increased in amount when we compare the age period 15 to 19 years with that of 10 to 14 years the difference is much more marked when the earlier age periods are taken into consideration. Much the same thing happens when the notifications

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received by the M.O.H of any large town are analysed but here I should prefer to take my statistics after the cases have been re-examined by the Tuberculosis Officer or other more special officer because it is a well known fact amongst such officers, that a great number of the children brought to them as reported cases of Active Pulmonary Tubercle are found on examination to be not such at all. To my mind the inference to be drawn from these facts is that the disease frequently exists in a form which is not easily diagnosed by ordinary clinical means or otherwise, that the disease is much less prevalent and also fatal amongst children. I am strongly inclined to think that the former is more likely to be the case. For diagnostic purposes phthisis pulmonalis in children may be divided into two types; first that in which the physical signs approach in character those observed in adult cases, and secondly a type in which the physical signs are obscure and ill-defined. As regards the first type I think the evidence goes to show that the class consists largely of "Contacts" i.e. patients who have contracted the disease direct from other patients. Halliday Sutherland⁽⁴⁾ gives the results of the examination of 723 contacts, of these 290 were found to be suffering from pulmonary tuberculosis 13 had other forms of the disease, and of the total number 372 were under 15 years of age.

From these figures and from clinical observation one can conclude that the type of phthisis pulmonalis which can be definitely diagnosed from the symptoms and physical signs alone is most frequently met with amongst contacts.

With a clear recognition of this fact a great stride has been made towards the early recognition of the disease amongst children. When a case of phthisis pulmonalis in the infectious stage is diagnosed, the matter should not be allowed to rest there especially if the patient is the parent of a family, or has come from a house in which there are also children residing. If amongst the children any are found to be delicate, either from apparent inanition or because of frequent attacks of bronchitis or so called "Colds", then a careful examination of these individuals by every possible means is certainly warranted. Ofcourse it may be argued by some that this is more a matter for the health authorities and their officers to attend to after the first notification, personally I do not agree with this and hold that it is distinctly within the province of the family practitioner. At any rate the converse holds good, namely that when dealing with a lung affection in the case of a child and which after a reasonable time does not definitely clear up, further enquiries

as to the environment of that child should certainly be made if they have not already been made; an adult case of phthisis in the house or lately in the house may often be traced, ~~this~~ throwing a strong light upon the possible diagnosis. I mention in the appendix an interesting case bearing upon these facts. When we come to deal with those cases of obscure latent phthisis pulmonalis amongst non-contacts in children, then a good deal more elucidation regarding the pathology of these will be required before ordinary diagnostic methods will lead one into supposing at once that they might be cases of tubercle.

Many authorities are inclined to believe that such cases are bovine in origin, without going into any academic discussion on this point, the conclusions of Fraser⁽⁸⁾ with regard to the incidence of bovine tuberculosis in children are interesting. He showed that a very considerable proportion of non-pulmonary tuberculosis in children is certainly bovine in origin. His work and also ^{the} consideration of the evidence of the Royal Commission on Tuberculosis⁽⁹⁾ of necessity compel one to come to the conclusion that a very definite proportion of cases of tuberculosis in children is due to the bovine type of bacillus. The R.C. showed

Royal Commission

that when the disease was generalised the human type of bacillus was usually the ~~caused~~ agent; at the same time the evidence suggested that the child was certainly more resistant to bovine tubercle than to human. It thus becomes possible that the slowly progressing obscure type of the disease, so often present in childhood and yet so difficult to definitely diagnose, is in many cases if not all of bovine origin. The patients of this class are usually the ones to which belong those, who present the classical stigmata of tuberculous infection.

I have already previously alluded to these as those whom our clinical forefathers classed as being possessed of the "Tubercular Diathesis". Thus ^{they} ~~it~~ may be phthisical in appearance without their chests yielding much definite evidence otherwise; they often possess a luxuriant growth of fine hair on the back. The lymphatic glands in the neck and axilla are often somewhat enlarged, whilst dilated veins may be observed coursing over the chest; behind the capillary veins over the spines of the upper three or four dorsal vertebrae may be congested. To this venous condition Lombardi⁽¹⁰⁾ gave the name of "The Varicose Zone of Alarm" and ascribed the phenomena as an important aid in the diagnosis of early apical phthisis. He thought that the

most probable cause of the condition was compression of the veins draining the upper intercostal spaces by pleural thickenings so commonly associated with apical phthisis. Lombardi claimed that this ~~varicose~~ condition was present in 90% of cases of primary tuberculosis of the apex. Others again and I think more correctly, ascribe this condition as due to compression of the superior intercostal veins by enlarged mediastinal (tracheo-bronchial) glands, certainly when this varicose condition does occur it is almost invariably associated with enlargement of these glands. This question and also the diagnosis of enlarged mediastinal glands, has already been discussed. More fully under "Physical Signs" (v. pp 41 & 103)

Another sign sometimes to be noticed in such cases is tenderness over the dorsal spines on firm pressure, ^{Spinalgia}. This may be a referred pain produced by irritation of the mediastinal nerves by pressure of the enlarged glands.

Petruschky⁽¹¹⁾ gave the probable causes of this condition as due to disturbances of the collateral circulation, to periosteal irritation, or to the presence of small tuberculous foci within the spine. Noeggerath and Salle⁽¹²⁾ also found hyperalgesia in the region of the fourth cervical to the second or third dorsal vertebrae (Head's Areas) and regarded this as due to early

tuberculosis of the bronchial glands.

Careful examination may reveal some degree of impaired resonance to percussion, or to combined percussion and auscultation, in some part of the lungs. The vibrating tuning fork along with auscultation may possibly reveal some abnormal area of consolidation as mentioned before in the diagnosis of enlarged mediastinal glands. It is very rarely indeed that the stethoscope alone reveals any adventitious sounds. Cough is often absent or it may appear at night and be absent during the day. The intermittent paroxysmal attacks of coughing may come on, which as mentioned before is often so like whooping cough. There is no sputum with such cases; occasionally there are attacks of dyspnoea at night, sometimes very like Asthma which may possibly be produced by irritation of the vagus nerve endings or possibly again by direct pressure of enlarged glands on the trachea (See Case xix). According to French,⁽¹³⁾ the chronic cough which is often met with in the case of town children is due to irritation of the right phrenic nerve.

The temperature curve may show nothing abnormal, occasionally the temperature in such cases is inclined to be regularly subnormal.

Any of the above signs or symptoms may be met

with in that innominate class of cases, tubercular in origin, to which I am referring at present. In some there may be more signs than others; it will be very rarely that all are found. Thus it will be seen that it is precisely to this class that the term "Suspects" can be truly applied, in other words the diagnosis is really a doubtful one if clinical means are relied upon alone. In no other class of case

are the more modern aids to diagnosis of more assistance than in this, they often give one a great amount of real help in estimating whether any particular case can be regarded as one of active tubercle. X-Ray examination usually reveals the position and extent of the lesions, whilst careful observation of the subcutaneous tuberculin reaction and a series of opsonic indices will largely assist in deciding upon the degree of activity of the disease. Lately Overend⁽¹⁶⁾ has strongly emphasised the value of X-Rays in the diagnosis of many cases of pulmonary tuberculosis in children.

The co-existence of five conditions which he gives should induce the school medical officer to examine carefully for pulmonary tuberculosis and enlarged bronchial glands. If in doubt the patient should be sent to the radiologist. These five conditions are as follows:—

1. Intermittent sudden cough in the absence of cases of Pertussis.
2. Washing, pallor and tiredness.
3. A sub-febrile temperature rising occasionally to 100°.
4. The presence of capillary veins, and a growth of hair over the upper thoracic spines.
5. The absence of any obvious disease.

To my mind, as said before, it is not the diagnosis of tubercle that is so difficult in some cases, but the estimation as to whether the disease is merely a latent one in a certain type of subject, or that early active disease is becoming superimposed on the old lesion.

In spite of all that has been said however, one often finds that after entertaining the gravest doubts about a given case the patient takes a turn for the better and after a short while is going about again as if there had been nothing serious wrong at all. Such cases as these are apt to make one exceedingly sceptical regarding the practical value of all methods of diagnosis when dealing with a supposedly tuberculous child in fact they often make one despondent regarding the value of one's real diagnostic powers. I am inclined to think that one or two experiences of this kind are not without a great deal of value to the clinician and teach him much more

about the practical application and the comparative value of all diagnostic methods, whether these be ancient or modern, than could be learnt from months of study from text-books, and that after all although we may ultimately diagnose correctly it is only in a slow stumbling and halting manner with many cases. All of this goes to prove the truth of my argument introduced at the beginning of this thesis, namely that the newer methods now often adopted for the diagnosis of pulmonary tuberculosis still require much further study and elucidation before their true value can be correctly determined.

Finally however when all the evidence has been sifted and weighed I am strongly of the opinion that if there is doubt, the patient should get the benefit of that doubt, namely the physician should err on the safe side and treat the child as if it were suffering from active disease. Such a course even if not absolutely necessary in some cases, will result with others in enormous benefit to them, if the supposition that the disease is active should really be correct. Even if the supposition is not correct, a great deal will have been done in strengthening the immunity of a child which has certainly at any rate given occasion for thinking that the

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already existing immunity was showing signs of
breaking down.

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SUMMARY & CONCLUSION.

"Reasons seen from afar seem to limit our view
but when we approach then we begin to see
beyond. ("Pascal")

I have now come to what I consider as perhaps the most difficult part of this thesis. One might naturally expect that after discussing, at such length, the signs and symptoms of phthisis pulmonalis I should now tabulate a formula for the quick and certain diagnosis of the disease in its incipient stages. Alas this is not possible, as a careful review of which I have written will show that no one method will suffice alone, for the diagnosis of all cases. I will even go so far as to state, that the combined use of all the available methods of to-day will not result in certain diagnosis of all early cases. Certainly many of them will be so diagnosed but there are a few about which the most that can be said is, that they raise a shrewd suspicion.

These should be treated as actual cases, because when they do become diagnosed with certainty, they are then probably no longer early cases, the mere fact of their being now capable of certain recognition proving that the disease has advanced.

If one were a strict adherent to the "No diagnosis, no treatment school, then I am afraid Tuberculosis would never be eradicated from our civilisation." Often in my musings I can see an array of past patients, all adolescents of the type to which I now refer. With them all the signs were negative yet their symptoms raised suspicion. In all, there was failure in the general health, some complained of loss of weight, others of slight cough, then again there were the few whose chief complaint apart from the failure in their general health lay in attacks of palpitation or a frequent feeling of throbbing in the temples, some had no additional symptoms except attacks of cold perspiration at night which were attributed to weakness, others had slight bleedings from the mouth which they were certain had their origin in the teeth or gums. Each patient presented one or other of these additional symptoms in a form or forms upon which I dwelt in greater length in the introduction to the subject. The common feature to all these cases practically devoid of physical signs, was the lowering of the general health, but only a moment's reflection will convince one that this symptom alone could not warrant one in coming to a diagnosis of incipient

phthisis pulmonalis in every such case, but yet when taken with the additional features to this, as mentioned, such a suspicion, and even then could not be called more than a strong suspicion, could not be put out of one's mind in each individual case. Retrospect as I have said in spite of all caution and due care on my part has proved to me that subsequently many of these showed the truth of the possible diagnosis to Treating accordingly does not necessarily mean that in all cases one must give the possible diagnosis to the patient, tactful advice regarding the rational treatment of the present condition may often be so given that in any case no ground may be lost owing to any delay in arriving at a certain diagnosis. Certainly there are a few patients who will positively demand to know what is at the back of the physician's mind, personally, my rule with these even at the risk of being called an alarmist is to tell them the truth, namely that I am not sure myself of the definite cause of the condition but think it may possibly be Consumption. Those who have taken actual offence at my opinion expressed so, have been remarkably few, ^{and have afforded me consolation as a rule} in the fact of reading their obituary notices in the newspaper may be about six months after my examining them.

Under the consideration of the physical signs I have held that percussion gives the earliest evidence in this respect. I know that most authorities do not accept this view but there are also a few good ones who do, many opponents to this view I think beg the question, and beg it wrongly when they misinterpret Osler to support them in saying that it is folly to expect dullness as an early sign, I do not expect dullness as an early sign, no real advocate of percussion whom I have ever met expects dullness as an early sign, a departure from the normal resonance, as estimated in early cases by comparing with the other side, is the sign looked for by percussion, which is a very different thing from saying that there is a dullness present over a certain area. As soon as physiological functions are interfered with, pathology commences, surely the physiological functions of any part of the lung is in the first place to oxygenate as completely as possible that quantity of venous blood which circulates through it. Now tuberculosis in its earliest stage essentially consists of a process of deposition and the first interference that this pathological condition could possibly produce would be in the direction of altering the

the prime physiological function of the organ as stated, that is, it would diminish that normal amount of tidal air entering the lobe of the organ first affected, a factor which *any* student of physics will affirm must cause an alteration in the resonance because of the normal elasticity of the organ.

The advocates of auscultation hold that this diminution in tidal capacity of the lung is first revealed by the stethoscope in the direction of diminishing the respiratory murmur. Now the stethoscope if it should happen to be placed exactly over the seat of the early lesion which is exceedingly problematical, may reveal evidence of diminished air entry to the part under examination and we have a lessening in the intensity of the normal respiratory sounds. On the other hand it may very likely be that the lesion is comparatively speaking a long way from the surface and that is also from the seat of the stethoscope, in other words the lesion may be stated in the centre of the lobe so that the first alteration to the respiratory murmur may be impossible of detection by the stethoscope. To sum this up then, a lesion in any part of a lobe must alter the air tension therefore the percussion note over the whole of that lobe; this

I have started

is a certainty, whereas the diminished ~~air~~ entry into any particular part of the lobe to a sufficient degree to allow of its detection by auscultation, can only be a matter of chance depending on the circumstances.

To my mind a common mistake is made by a great many who have the idea that alteration in the percussion ~~hole~~ in phthisis pulmonalis depends on the occurrence of a generalised consolidating fibrotic process throughout a large part of the lung, this of ~~course~~ is comparatively a very late process. This is not the cause of diminished resonance and alteration in the percussion ~~hole~~ this as I have said, depends on a diminution in the air tension of the whole of the affected lobe, which for reasons based on physiological principles, is the first alteration from the normal function which commencing disease could possibly produce. I certainly cannot agree with those who hold that a ^{few} fine crepitations heard at the end of the inspiration after coughing is the earliest sign of the disease. Certainly it is far from unusual to find these in a patient coming up for his first consultation but the reason for this is that the patient has either disregarded for some cause or other the first symptoms of his affection, or else if

he did ~~not~~ consult a medical man on an earlier occasion, the latter has failed to recognise the possible significance of his symptoms. Isolated crepitations indicate that the pathological processes have advanced a step further and that already a breaking down of lung tissue is beginning to take place.

I need say little more about the evidence derived from Radiography, Tuberculin Tests.

and the Laboratory; at present they all can only be classed as adjuvants to the clinical evidence, and cannot in themselves alone allow of the detection of incipient active disease.

The more they are properly considered the clearer their position stands out in the diagnostic view; they are sometimes good guide posts, especially to the young and inexperienced practitioner, on a difficult cross-tracked road, but this is the most that can be said of them; the road to diagnosis itself, is essentially the clinical evidence, and to those who have trained themselves to appreciate its many and fickle manifestations it becomes clearer and clearer, so clear in fact that by and by the experienced clinician rarely has to glance up to these posts for his direction. How rarely are cases of incipient phthisis pulmonalis coming under the notice of our old clinical teachers missed, yet they never with-

hold a diagnosis because forsooth the reports from the pathologist or chemist and so on have not yet arrived. It is most important that such time-taking evidence should not be allowed to withhold correct treatment from the patient so that as Prof. Lindsay has so eloquently put it "While we examine blood sputum urine, gastric contents and, let us not forget at the same time to examine the patient. "Neglect of this important principle may lead us into exactly the same kind of mistake as the ancients "Deliberat Roma," perit Saguntum," rather must it be as Juvenal has aptly put it "Nulla unquam de morte hominis cunctatio longa est."

It must never be forgotten that next to prevention, early diagnosis takes the highest place of importance in the campaign against tuberculosis. Prevention for the tubercular, early diagnosis for the tuberculous, note the distinction.

I am now firmly of the opinion that the healthy individuals of civilised European races are mostly all tuberculised, meaning thereby that they possess, in virtue of having hitherto resisted the almost omni-present tubercle bacillus an acquired immunity of greater or lesser extent to its further ravages.

As commonly understood by the term in the consideration of tuberculosis I deny the existence

of a pretubercular state. From the weaning period of the infant right up to old age freedom from consumption depends on the balance struck between the toxin and the antitoxin or immune-bodies present in the circulating blood. How often are patients informed that they have not got consumption but that they are seriously threatened with it, or again that their vitality is in such a low state that the germ of tuberculosis is about to attack them, as if the T.B. had not been attacking them all their lives, and usually to the host's advantage at that.

Freedom from active tuberculosis, depends I say on a good balance, of life long standing, of antitoxin over toxin, i.e. that the body cells have nobly responded to the stimulating toxin by the production of immune-bodies in far more than sufficient amount to overcome it.

The so called "Tubercular Diathesis," I use the term in a descriptive sense for want of better is the result of a long continued hard fought battle in the child between the toxin and antitoxin generated, in which the body has not escaped from certain physical results of the fray, but has on the whole up to the present come out on the right side. With such a case the immune-bodies have been generated in just rather more than sufficient quantity only, i.e. the body

cells have given a relatively poor response to the call upon them for antitoxin by the toxin. The due appreciation of these two types of cases renders the understanding of the others clearer, whilst at the same time it helps considerably in the practical application of the diagnostic methods already discussed. Thus for example there is that large class of cases to which I have often referred in this thesis, and who are a constant source of difficulty when the more special methods of diagnosis come to be applied. I refer to those adults who exhibit the characteristic stigmata of hard earned though successful resistance, the lesions are not active they are latent and usually associated with a large amount of fibrotic consolidation of the posterior mediastinal glands. These individuals possess a high degree of sensitiveness to the products of the tubercle bacillus i.e. tuberculin. Often enough these patients belong to that class who as children were said to be threatened with consumption in some part of the body, their degree of immunity has increased *pari passu* with their age until at the stage of adolescence, although they cannot be called robust looking individuals are certainly not unhealthy. All the lives the struggle between infection and the immune bodies has been going on, the latter however at last coming out victorious. I can safely

say that hosts of this class have on the slightest provocation been diagnosed as victims of active phthisis pulmonalis and have also, almost as often been shown as examples of the cured disease under some new form of treatment or other. Such a state of affairs is neither just to the patient nor to the public, it vitiates statistics and tends to bias a true judgment when an estimate of the progress of the campaign against the disease is being considered. These are the patients who are apt to be swept wholesale into the net when the special aids to diagnosis are depended upon more than careful consideration of all the circumstances should warrant. It cannot be denied however that many of this class do actually break down at times and for the time being the balance between immunisation and infection has been overcome, but it is my belief that in the great majority of such instances these cases do very well and make a splendid recovery under no special form of treatment except rest and good hygienic surroundings. My point however is that unless the clinical evidence is very carefully weighed, and if at the same time it should happen that too much attention should be paid to the results of the tests by tuberculin X-Rays, and so on, many of these patients will

be diagnosed as having become actively tubercular when in reality they have not. One is exceedingly apt to forget that such subjects at the same time can also suffer from the other diseases which flesh is heir to. When I said that prevention was best, the remark must be very specially applied to these individuals, who for brevity may be said to be in a healthy latent stage of tuberculosis. Early diagnosis of an actual break down is next best for these persons, always being exceedingly careful however to be actually sure that such a state of affairs has arisen before labelling such a person for the first time in his life as a victim of phthisis pulmonalis, indeed the great difficulty with such cases is for the clinician to refrain from making an error of commission, whereas ^{with} most of the difficult cases for diagnosis the making of an error of omission is what the clinician has most to guard against. I have dwelt a great deal upon this class of case but my excuse must be, lest like Horace "Brevis esse laboro, obscurus fio".

How now must the hitherto healthy adolescent contracting the disease be considered? The theory that I have advocated supposed that such was possessed of a large amount of immune bodies stimulated by successful resistance to the

tubercular toxin i. e. there is a large degree of active acquired immunity. At first sight one would think that if this theory were correct, it would be impossible for such a person to contract the disease. The degree of immunity which any individual possess against any particular disease is as well as known, dependant to ~~the~~^a large extent on certain well defined factors, such for example are cold, fatigue, poor food, bad housing sanitation, and lastly though by no means of least importance, the existence of other diseases. All these factors which I have already dwelt upon under the consideration of the history of the illness, are precisely ~~called~~^{those} which have a destructive effect on the immune-bodies, and which I have previously called the chief predisposing or contributory factors to the disease. Does not clinical evidence as well as consideration of the opsonic index demonstrate how apt the disease is to set in after Measles, Whooping-Cough, Influenza, and Syphilis. The tubercle bacillus flourishes on its host when it lives in the ~~sympiotic~~^{ie} condition with other germs, simply because the micro-organisms of these other diseases or their toxins have had the effect of neutralising or exhausting the immune-bodies previously present, it is as if a strong brake had been

applied to the active processes of phagocytosis. Thus it is, that the too zealous devotees at the shrines of Bacchus Vulcan and Venus often fall victims to the disease.

Gradually immunisation succumbs to grosser infection and gradually there sets in that state of health which is often called the pre-tuberculous state. This is a term I strongly object to, ^amisnomer when applied to cases like this. What happens is that gradually the antitoxin becomes too weak to successfully resist and hold in check the omni-present toxin, which thus brings about in the host those symptoms which are often classed under this term of pre-tuberculous state, or otherwise that the patient is threatened with consumption. It is not a case of being only threatened with the disease at all, it is actually the first manifestation of active tubercule setting in, the only difficulty that exists is the correct interpretation in every case of the cause of such symptoms, because such are identical with the early symptoms of many other less serious diseases. If loss of weight and bone then is the first manifestation of active tubercule in these cases then certainly it is folly to call such a condition a predisposing factor only. The moral to be drawn from this then is that when we have to deal with manifestations

which may possibly be the ^{first} stage of active tubercle, this possibility must not be lost sight of for a moment, until a definite declaration regarding the case is possible, whilst the treatment to be adopted from the first must be such that is not incompatible with the correct management of actual early tubercle.

The case of the infant contracting acute tubercle has now to be considered under the light of this theory. It is rightly said that it is at present impossible to diagnose early tubercle in an infant; the reason for this is that in the infant, tuberculosis is usually an extremely rapid disease passing quickly to a fatal termination, or against if it should happen that suspicions of the disease being active tuberculosis are at first held, if the infant recovers then one assumes that such suspicions were wrong. There is nothing strange in this when it is considered that the human bacillus gaining an entry into the body of an infant has practically virgin soil to flourish in. Here immune-bodies have not had time to become generated under the benign influence of repeated small doses of toxin, such only occurs with increasing age of the infant and then probably under the stimulating influence of the much less virulent bovine type of bacillus. Exactly the same occurs when the white man's scourge is

introduced first, amongst a native population. When I first arrived in the West Indies, over and over again I was surprised at the great number of cases of apparently epidemic acute-pneumonia which proceeded to a fatal termination after about five to seven weeks illness. Post-Mortem examination of many of these cases confirmed to me the fact that active tuberculosis was the cause of death. The natives although possessed of a certain degree of natural immunity when infected, have not as yet attained any marked degree of acquired immunity analogous to that of the European races. The reason for this probably is, that tuberculosis is a much older disease amongst the latter than the former. Many consumptives, more particularly Americans from the Eastern States are now finding their way to the West Indian Islands for the benefit of their health, the climate being in many respects an ideal one for such patients. That the disease is really not more prevented than it is amongst the natives must be set down as due to their natural immunity only, and also to their practically almost continuous out-door life in such an excellent climate. I do not remember of ever having seen a case of chronic fibroid phthisis in a West Indian native, or if I did I missed the diagnosis of the condition.

My last remarks must be directed towards the duty of the patient if the real early cases are to be obtained in any quantity. Well has Hippocrates affirmed in his first aphorism that it is not only sufficient for the physician to do his part but that the patient also has a great duty to perform. How often does one meet with the case where, had the patient reported himself sooner the prognosis would probably have been an excellent one. This most important factor towards rendering an early diagnosis possible is often forgotten, and I am afraid that a quick diagnosis by the physician is often far from being synonymous with an early one for the patient "*Facilis decensus Averno*".

"*Sed remocare gradus superasque evadere ad auras. Hoc apus, hic labor est.*" It becomes essential therefore that the public intelligence should be aroused to a correct appreciation of this fact, no matter how skilled the physician may be in diagnostic methods, if the public are not educated upon such matters as the common factors causing the spread of the disease and the dangers which contracts have to guard against, then I am afraid that a large number, and probably the great majority of the real early cases, will be missed. The physician should not wait for the contracts to come to him, he should go to the contracts. The history

and records of every tuberculosis dispensary in the land will vouch for the truth of these statements. How ~~morn~~ful is it to hear of such a statement as the following from the lips of no less an authority than Sir. A. Newsholme

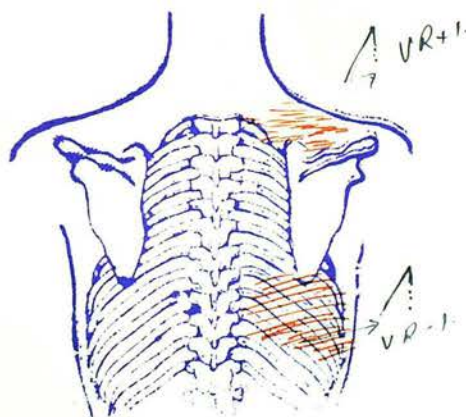
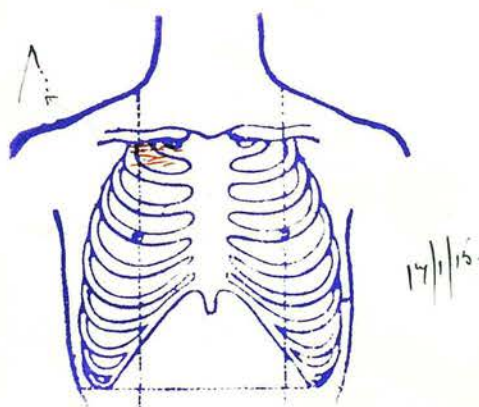
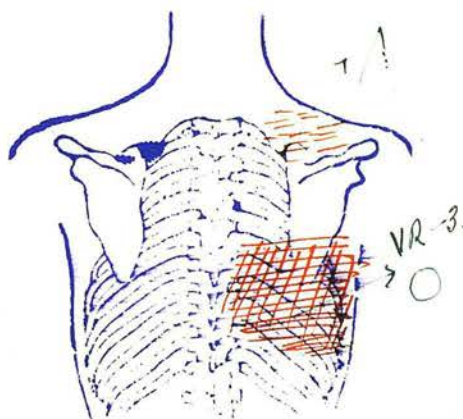
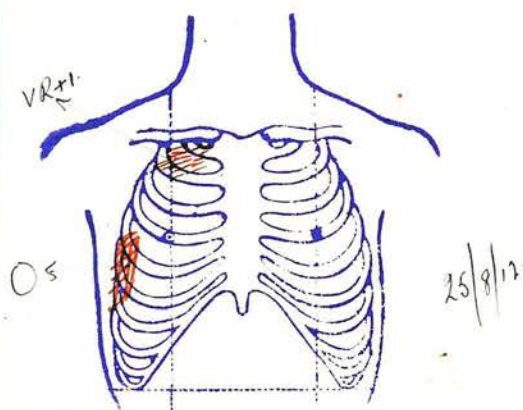
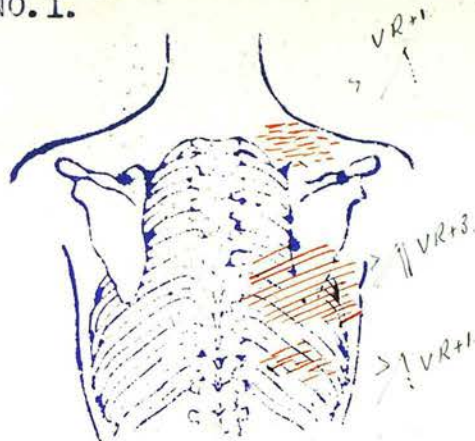
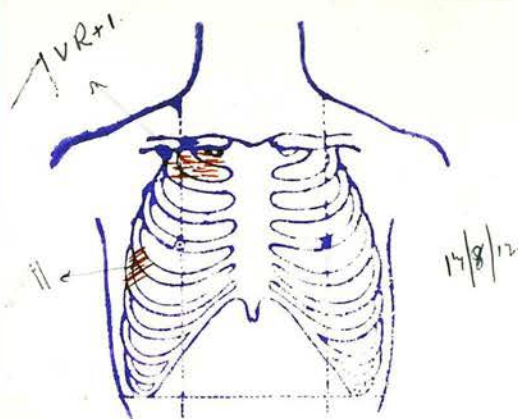
"That under present conditions a large proportion of the total cases of phthisis pulmonalis remain unrecognised until either consolidation or cavitation of the lungs has occurred. *Thus it is that while tuberculosis has often been described as a disease of misery it can be much more truly called a disease of ignorance. I can only pray that the time now is, or will shortly be, when it will become impossible for the general medical practitioner to share in the slightest degree with the public the great weight of responsibility for such an awful state of affairs as this. For such a medical utopia to arise will depend on much the same principle as the successful treatment of phthisis pulmonalis depends, namely upon the early training of the general medical practitioner i. e. whilst he is still a student the essential principles in the diagnosis and treatment of early pulmonary tuberculosis cannot be too forcibly brought home to him, also

the tragic train of events which are likely to happen should neglect of these principles occur must be clearly demonstrated. The mind of

every young graduate is perfectly clear upon the importance of the early diagnosis of malignant disease. I am not quite sure however that in every case it is as perfectly clear upon the early diagnosis of phthisis pulmonalis and how an early case ought to be defined. I know that my own alma mater is, and rightly so a Spartan mother, this matter and if any of her alumni do fail in these respects it is through no lack of teaching and warning on her part. Foremost amongst the banners carried by the stalwarts in the crusade against tuberculosis we can see two, towering above all the others, one emblazoned with the word "Prevention", the other with "Early Diagnosis". The former it may be observed is directed more towards the public in general, while the latter is more inclined towards the medical profession, nevertheless both are slowly but surely becoming plainer and plainer for the whole world to see. If we draw closer to the cavalcade and come to examine the symbols on the streamers linking these two banners together, we can read as follows in letters all of gold, those well known lines of Shakespeare nowhere to be found more aptly applied.

"There is a tide in the affairs of man which taken at the flood leads into fortune omitted all the voyage of their lives is bound in

shallows and in miseries".



H.G. Age 43 years. Housewife. Admitted to
Monkwearmouth Hospital in August 1912. Complained
of pain in the side and cough. T.104. P.108.

CASE NO.1 (Continued).

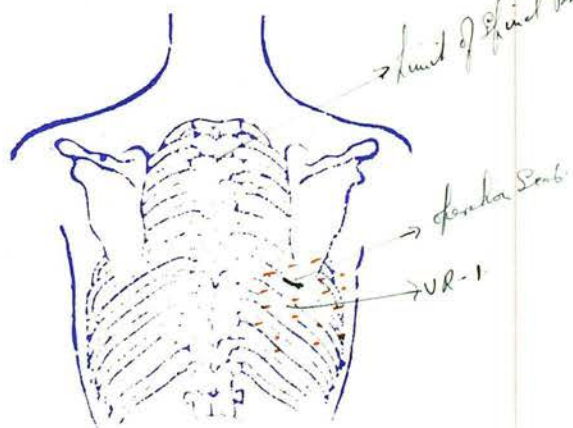
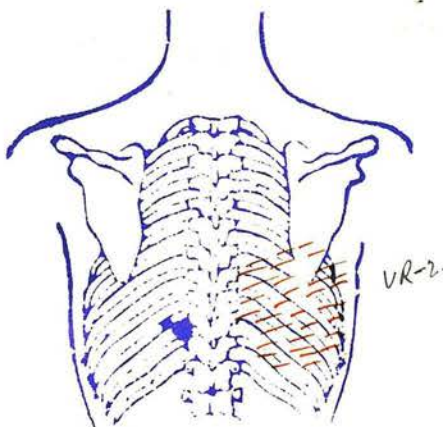
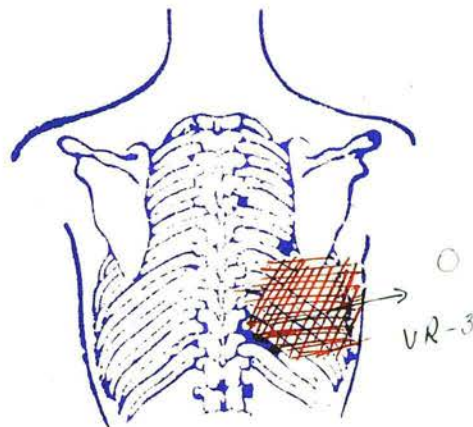
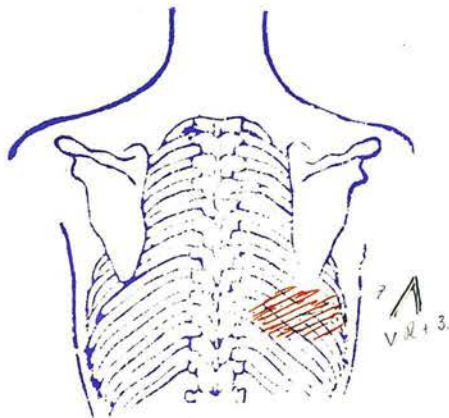
She was thin and emaciated looking woman. On examination she was found to be suffering from an Acute Pneumonia affecting the middle and lower lobes of the right lung. Her crisis occurred on the third day after admission.

The patient did not seem to recover well, whilst she kept on coughing a great deal. On examination of her chest again about one week later, the dullness over the right side of her back was found now to be absolute; respiratory movements were also very shallow over this area. Exploratory puncture revealed the presence of a thin yellowish fluid, about 24 ozs. of which was at once drawn off. She did not require to be tapped, again, whilst she made good progress towards recovery. She left the Hospital at the end of September looking and feeling very much better; there was still a large amount of dullness at the right base which was probably due to pleural thickening. Her weight was 7 st. 10 lbs.

I examined the patient again in January 1915. She had kept very fair health since leaving Hospital, and was looking very much stouter than when I saw her last, her weight now being 8 st. 7 lbs.

(216)
CASE. NO. 2.

H.L. Age 7 years. Admitted to Monkwearmouth Hospital at the end of September 1913 as a case of Acute Pneumonia in its sixth day. T.101. P.113.



→ kind of Spinal Bronchopneum.

→ dorsal Sub.

→ VR-1

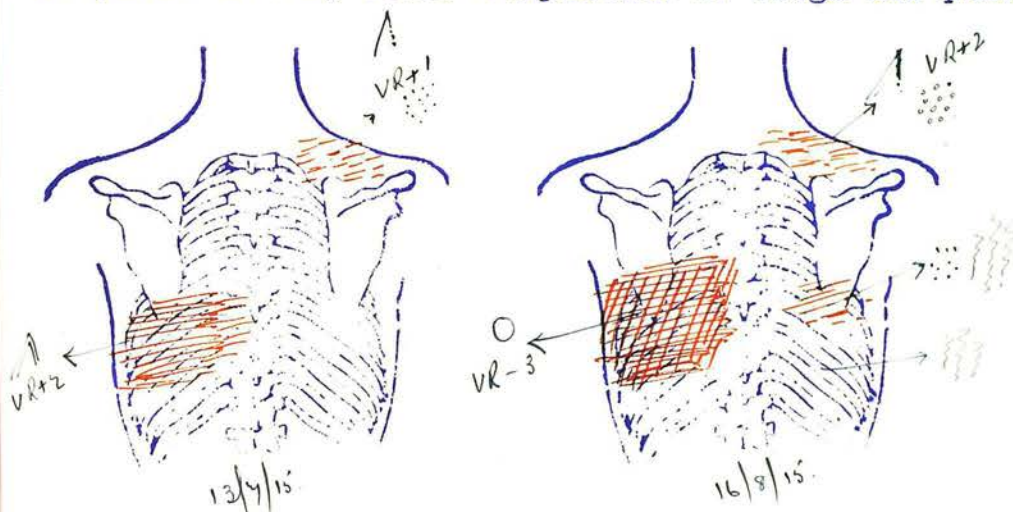
The attack had come on suddenly in a child which had always been fairly healthy. On examination a small patch of dullness with tubular breathing to be heard over it, was found near the right base. There was no definite crisis and the child did not show any great signs towards recovery after one week. Examination now showed the dullness to have

extended all over the base behind, whilst it was now also absolute. Exploratory puncture revealed the presence of a fluid which though not quite was approaching the nature of pus. The patient was handed over to the Surgeon (Dr. Gilbertson), who resected a portion of the 7th. rib in the posterior scapular line which allowed of free drainage.

Improvement was rapid after this and the child was discharged in the beginning of December 1913. Subsequent examination in August 1914 showed the child to be in a quite healthy condition; there was just a little comparative dullness around the region of the operation scar. Spinal bronchophony was distinctly heard as far down as the upper part of the 3rd. D.V.; the intercostal veins were fairly distinct and prominent in the upper part of the right chest in front. These signs probably indicated a lesion, probably now quiescent in the posterior mediastinal glands.

CASE. NO. 3.

T.C. Age 47 years. Miner. Admitted to Monkwearmouth Hospital in July 1913. Complained of cough and pain



in the side. His illness had started with a shivering fit five days previously. Three years ago he had been told by his doctor that he was threatened with consumption since which time he had been troubled with a cough off and on ever since, whilst every now and again he had been laid up for a few days with a "Cold".

The present was the worst attack he had ever had. On examination the patient was found to be thin and emaciated looking, whilst over the left base behind a large patch of dullness having tubular breathing heard over it, was to be found.

Over the apex of the right lung behind a certain amount of comparative dullness was to be detected, and at the same time a few fine crepitations could be heard towards the end of inspiration and beginning of expiration. The sputum was found to be tenacious and rusty; pneumococci but no T.B. were

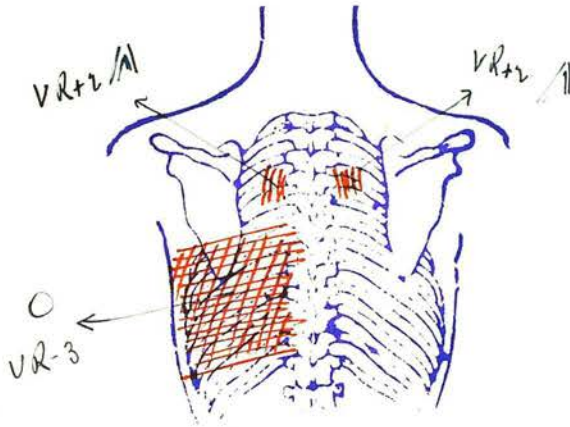
CASE. No. 3 (Continued.)
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found to be present.

The patient made no progress at all whilst the temperature began to take on a swinging character. Exploratory puncture made towards the end of July revealed the presence of a thin pus in the left thorax behind and below. The patient absolutely refused operation whilst attempted aspiration of the fluid yielded little success. He died on the 16th. of August 1913.

CASE. NO. 4.

H.W. Age 61 years. Plantation Worker. Married,
with eleven of a family. Admitted to the General



Hospital, Barbados.B.W.I. in March 1906.

She complained of great weakness, shortness of breath, cough and attacks of vomiting at times. On examination the patient was found to be very much emaciated. The sputum was copious and very dark coloured ie: it was typically Prune-juice like in character. The whole of the left half of the chest behind and below was found to be bulging and immobile a respiration; dullness over this area was absolute. Two patches of dullness one on each side of the spines of the 4th. and 5th. D.V. were also to be detected; there was no definite area of dullness to be detected in front.

Exploratory puncture showed the presence of a dark blood stained fluid, about 17 ozs. of which were drawn off. Sudden regurgitation of food took place on two occasions whilst she remained in the Institution. This occurred when an attempt was made to give her a more solid diet. In spite of

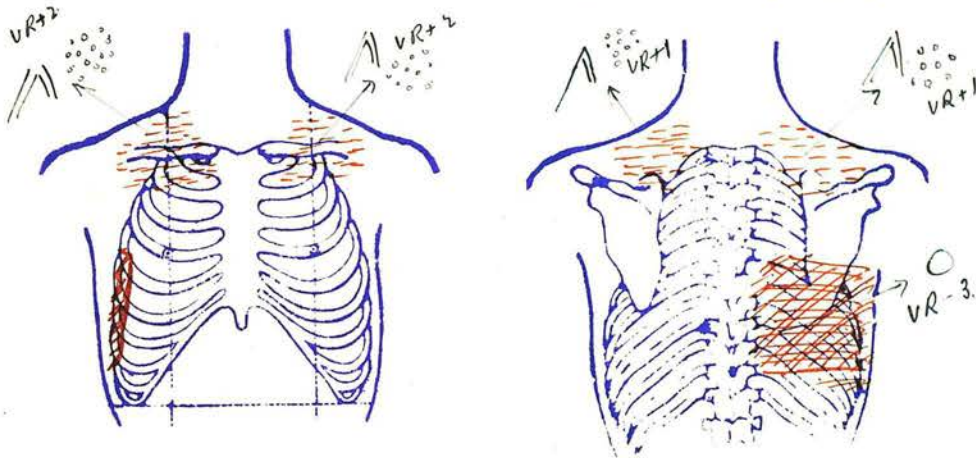
CASE. NO.4 (Continued.)

this however there did not appear to be any definite stoppage to an oesophageal bougie. She left the Hospital 14 days after admission and her subsequent history was unknown to me.

The diagnosis was Carcinoma of the left lung probably secondary to a primary growth at the lower end of the oesophagus.

(122)
CASE. NO. 5.

H.G. Age 22 years. Admitted from O.P. Department to Monkwearmouth Hospital December 9th. 1912 as a case of Pleurisy. Effusion. The history was,



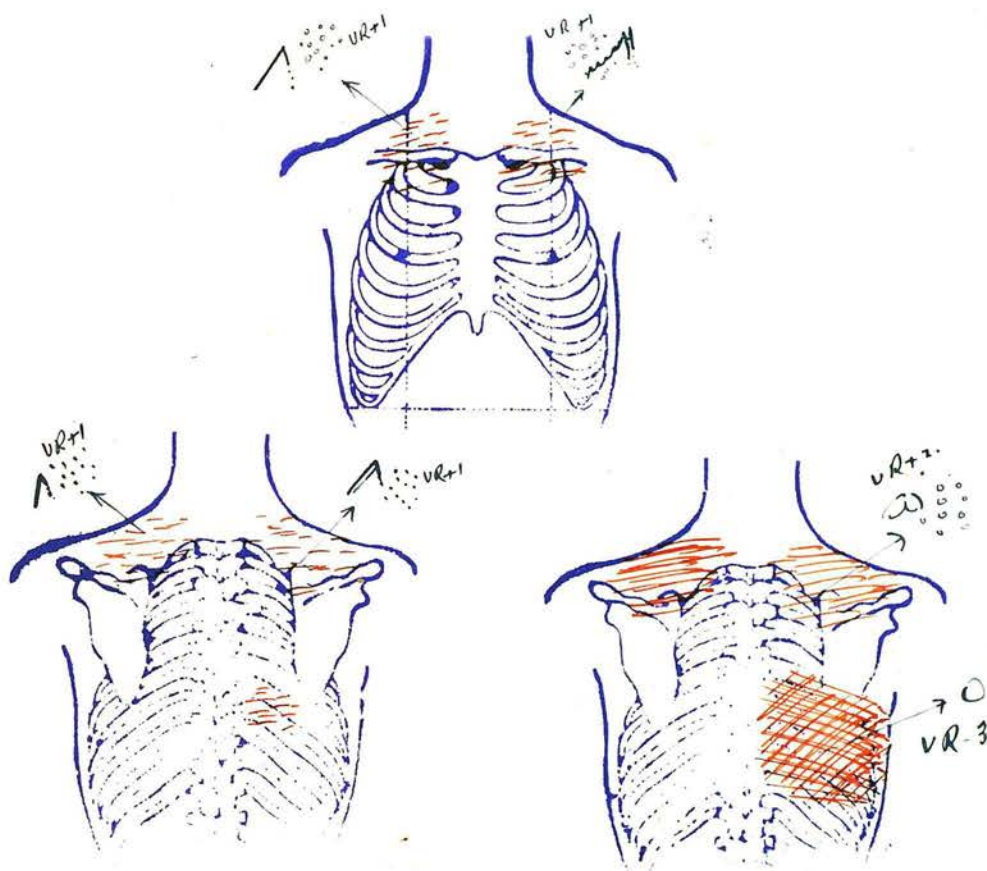
that she had been ill for about 18 months with cough, & shortness of breath; had been told by her own doctor that she was suffering from consumption. On examination there was absolute dullness over the lower chest on the right side behind extending into the axillar region. Dullness also over ~~the~~ both apices, both in front and behind.

Exploratory puncture confirmed the diagnosis of Pleurisy & Effusion. Sputum copious thick and tenacious, containing T.B.

As patient was coughing a good deal she was not deemed a suitable case for the Institution so she was discharged after 4 days.

(313)
CASE. NO. 6.

N.J. Age 18 yaers. Crook, Co. Durham. First came



under my notice in February 1910, suffering from loss of weight, cough and attacks of cold perspiration at night; had been ill for some months before my seeing him. On examination dullness found at both apices, both in front and behind; coarse crepitations with increase of V.R. heard in the infraclavicular ~~es~~ supraclavicular areas on both sides. Dullness and a few fine creps also to be made out at apex of middle lobe of the lung behind. Patient gradually got worse, sputum became abundant and blood specked in which numerous T.B. ^{were} eventually found, although not found at first. Shortmess of breath became very marked and on

CASE. NO. 6 (Continued.)

examination of the chest in May, marked dullness and immobility, with absence of breath sounds was to be observed at the lower part of right chest behind. Exploratory puncture confirmed presence of yellow fluid. Patient died shortly after this in the beginning of June.

CASE. NO. 7.

H.M. Age 25 years. Examined at O.P. Department of Monkwearmouth Hospital on July 10th. 1913. Complained of great shortness of breath and cough. Patient was aware that he was suffering from consumption having been informed of the fact by his doctor, fifteen months before.

On examination lower left half of chest behind was found dull and immobile, along with absence of respiratory sounds. Exploratory puncture revealed presence of fluid. Patient refused to come into the Institution.

CASE. NO. 8.

R.H. Age 21 years. Examined at Tuberculosis Dispensary in April 1914. Patient had been attending at the Institution weekly for about three weeks without much improvement. No effusion was noticed in the chest on the previous visit. On examination the lower right half of chest behind was found dull and immobile. Respiratory sounds not audible over this area. Exploratory puncture confirmed presence of fluid.

(226)
CASE. NO. 9.

P.G. Age 27 years. Examined at the Tuberculosis Dispensary in July 1914. Patient had been sent to the Institution as a case of Phthisis Pulmonalis for treatment. On examination dullness was found over lower half of left chest behind.

Respiratory movement here very feeble. No vocal resonance or breath sounds audible. Exploratory puncture revealed presence of fluid. Nature of the pleurisy revealed by the condition of the ^{apex} apex; here dullness and crepitations were early detected.

CASE. NO. 10.

J.W. Age 37 years. Examined at the S.R.D.I. Hospital in March 1913. Was admitted to the Institution for treatment on March 3rd. as a case of Phthisis Pulmonalis complicated by Pleurisy & Effusion. Examination revealed the presence of fluid in lower part of right chest behind; this was confirmed by exploratory puncture. The left apex revealed the presence of active mischief; the condition of the right was doubtful but probably also affected.

CASE. NO. 11.

J.B. Age 42 years. Examined in August 1914. at request of an Insurance Company.

There was a history of having sustained an accident to his back about 3 months before and for which compensation had been paid. On examination he was found to be a thin decrepit broken down man; a great deal of tenderness real or assumed was to be observed over the lower lumbar and upper sacral vertebrae.

In the lower half of left chest there was absolute dullness along with absence of V.R. and audible respiratory murmur. I was convinced of the presence of fluid here but owing to circumstances could not confirm the diagnosis by exploratory puncture. Examination of the urine very shortly after it being passed showed it to be acid in reaction; and very cloudy.

By the guaiac- ether test, blood was shown to be present which was also suspected from the appearance of the specimen; Examination of the deposit showed the presence of pus. To my mind pus and blood present in an acid urine is strong presumptive clinical evidence of tubercular disease of the kidney. The kidney was probably the primary source of infection to set up the Pleurisy & Effusion.

CASE. NO. 12.
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D.L. Age 27 years. Admitted to Monkwearmouth Hospital July 1913 suffering from Tubercular Disease of the Ankle-joint; as the joint was completely disorganised and the foot useless, amputation below the knee joint was performed by Dr. O'Flaherty, the H.S.,

The patient shortly after the operation was handed over to me because of his having developed chest trouble. On examination marked dullness at both apices behind was to be made out particularly so on the left side, breath sounds over these areas faint and interrupted in type. Over the right lower half of the chest there was bulging and absolute dullness to percussion along with absence of respiratory murmurs. Exploratory puncture revealed the presence of fluid about 26 ozs. of which was drawn off. Against our wishes the patient left the Hospital 8 days after this. He died about three weeks after leaving the Hospital as I was informed by Dr. Thompson, his private medical attendant.

CASE. NO. 13.
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J.H. Admitted to the S.R.D.I. Hospital in November 1913. as a case of Typhoid. In the absence of my chief, (Dr. R.Stobo) the patient was under my care. The history was that about four weeks previously the patient whilst working in the pit received a severe blow on the head from a fall of stone which had stunned him; he lay off work and seemed to recover fairly well although he never resumed work. A fortnight after the accident he began to be troubled with head-ache and pains about the joints and back along with cough; this continued for 10 to 12 days gradually getting worse whilst the patient became semi-comatose muttering and delirious at times, Typhoid was diagnosed by his medical attendant and the patient sent into the Isolation Hospital.

On examination the abdomen was found tumid and rather distended, the superficial abdominal reflexes on both sides were absent; there were no areas of tenderness and no rose spots to be discovered; the bowels were inclined to be confined whilst the stool after an enema was not typhoid like. The patient was semi-comatose being aroused with difficulty; breathing however was shallow and rather quick at times, if anything the left pupil was slightly larger than the right, both responded sluggishly to light. Lumbar puncture yielded a fluid only slightly turbid which on examination seemed to show a rather greater proportion of

CASE. NO.13 (Continued.)
=====

Lymphocytes than other cells. I was unable to find any T.B. or diplococci. Widal reaction was negative; this was confirmed by the bacteriologist at Newcastle School of Medicine (Dr. Hutchens.) Chest examination revealed absolute dullness at the left base extending upwards, there were râles and rhonchi all over the chest. Exploratory puncture revealed the presence of a turbid yellowish fluid not at all thick ~~however~~ in the left pleural cavity. Although I had no doubt in my own mind as to the cause of death yet in consideration of the history I communicated the facts to the Durham Co. Coroner (Mr. Cadle) who shortly after ordered me to make a P.M. examination, at the same time stating that he was going to hold an inquest.

P. M. examination at once showed that the patient had succumbed to a generalised acute tuberculosis. The peritoneal cavity contained about three or four pints of greenish fluid, whilst the peritoneum itself was studded ^{all} over with small caseous tubercles. The left lung was collapsed, whilst the pleural cavity was filled with a greenish yellowish fluid. On section of the lung a large cavity about the size of an egg was found in the lower ~~to be~~ near the base and posteriorly, which had ruptured into the pleural cavity; over the rest of both lungs there were numerous small caseating

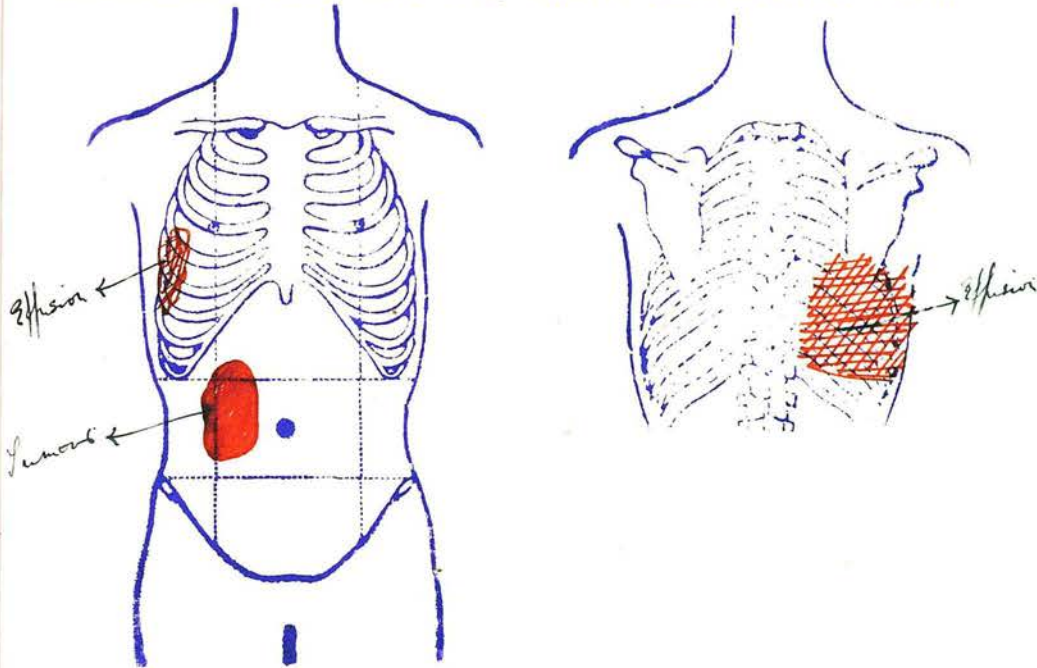
CASE .NO. 13 (Continued.)

tubercles.

The brain showed opalescence of the pia-arachnoid which was also studded with small tubercles; this was chiefly marked in the barrel region over the pons, extending backwards to the medulla and on to the cerebellum. Forwards it extended to the optic chiasm, into the anterior perforated spot and thence into the first part of the fissure of Sylvius.

CASE. NO. 14.

E. T. Age 37 years. Consulted me first in September 1913 when on a visit home from S. Africa. She complained of having a lump in her abdomen which was causing her a good deal of worry. She had not noticed this swelling until shortly after her arrival in this country, where she had now been



about 4 months. On examination one could feel an exceedingly tensile oval swelling about the size and shape of a large Jaffa orange occupying the right half of the umbilical and part of the right lumbar regions. The patient struck me as being rather neurotic in temperament, and this along with her statement that she had lost a good deal of weight lately made me incline to the diagnosis of a moveable kidney as the source of her trouble.

I advised her to adopt a fattening diet and also to wear a kidney belt. I saw her again six weeks after when she informed me she was not much

CASE. NO. 14 (Continued.)

better. I now advised her to see Mr. Turner of Newcastle with a view to operation which she did. Mr. Turner wrote me to the effect that whilst the possibility of an enlarged moveable kidney could not be overlooked, he was much more inclined to the view however that the swelling was a mass of enlarged mesenteric glands probably tubercular; he did not advise operation at present but would do so if the condition did not greatly improve under medical treatment during the following period of two months.

About three weeks after this I was called to see the patient, because she had contracted a chill. On my examination of her chest I found that she had a fairly extensive dry pleurisy over the lower right half of her chest; this was followed by a gradually increasing pleural effusion. I tapped her on two occasions at an interval of one week; each time drawing off about 17 ozs or 18 ozs. of clear fluid. She made gradual progress towards recovery and was able to get about again after an illness of nine weeks. She would not consider any question of operation now, and fully made up her mind to return to S. Africa as soon as she could as she was convinced that this climate did not suit her now. She left for home in April 1914 and since then I have had no further news of her.

I think the case rather an interesting one,

CASE. NO. 14 (Continued.)

the later history of the patient certainly warranted the diagnosis suggested by Mr. Turner, with which I certainly was induced to disagree at first.

CASE. NO. 15.

P. McC. Age 49 years. Seaman. Consulted me in October 1913; suffering from shortness of breath and cough which he said had troubled him now for about three months but during the last fortnight had got much worse. He admitted to having been a heavy drinker and to having suffered from Syphilis about 7 years ago; as far as I could judge however he seemed to have gone through a complete course of treatment for this. He attributed the onset of his present illness to a fall upon his back sustained about 3 months ago.

On examination he was found to be a thin broken down looking man, and the apex heart was found visible in the 5th. interspace but rather to the outside of the nipple line. The right border of the heart was out to the extent of about $\frac{3}{8}$ "; there was visible, though not marked pulsation of the veins of the neck. The liver was enlarged to the extent of about $1\frac{1}{2}$ " below the costal margin. I was of the opinion that this was real enlargement and not merely displacement downwards. There was just slight puffiness in the region of the ankle joints. The urine contained rather more than a trace of albumen whilst the patient did not think he was passing less than usual. On auscultation a loud systolic bruit could be heard in the mitral area. Behind, over the upper part of the left chest loud friction sounds could be detected. These

CASE. No. 15. (Continued.)

disappeared towards the left base where the breath sounds were entirely absent. On percussion here, absolute dullness could be detected. I needled the thorax over this area and confirmed the presence of fluid. The rest of his chest did not suggest anything particular apart from a little comparative dullness above the right clavicle in front; this I was inclined to think, in view of the ~~absence~~ presence of fluid in the left pleural cavity might have been more apparent than real.

Examination of his arteries did not suggest any pronounced degree of arterio-sclerosis.

The patient insisted on going home to London the same night and hence I could not complete my observations. I was inclined to think however that the case was fundamentally one of commencing Chronic Bright's Disease.

CASE. No. 16.

Q.R. Age 18 years. Laundry hand. Examined at O.P. Department of Monkwearmouth Hospital in February 1912. Complained of shortness of breath alongwith loss of appetite which she said had now being going on for about 7 weeks. Although anaemic she was plump looking and well built.

Examination revealed absolute dullness over the right base behind; there was also marked diminution of respiratory movements and vocal resonance. Nothing else abnormal could be detected in the chest. She refused to allow me to needle her or to admit her into the Institution.

Although not quite certain I was inclined to think that the case was one of Tuberculous Pleurisy c Effusion.

[Cases i. to xvi contribute the Pleurisy Series as mentioned in the text]

CASE. No. 17.

K.L. Southwick. Age 11 years. Came under my notice in private practice just over three years ago. I quote the case simply as ^{an} illustration of the voice changes apart from ulceration of the vocal cords which may occur in Phthisis Pulmonalis. This child is possessed of a deep voice sounding almost masculine, under excitement or much speaking the voice rapidly becomes hoarse and almost lost. This recovers again however in a very

CASE. No. 17. (Continued.)

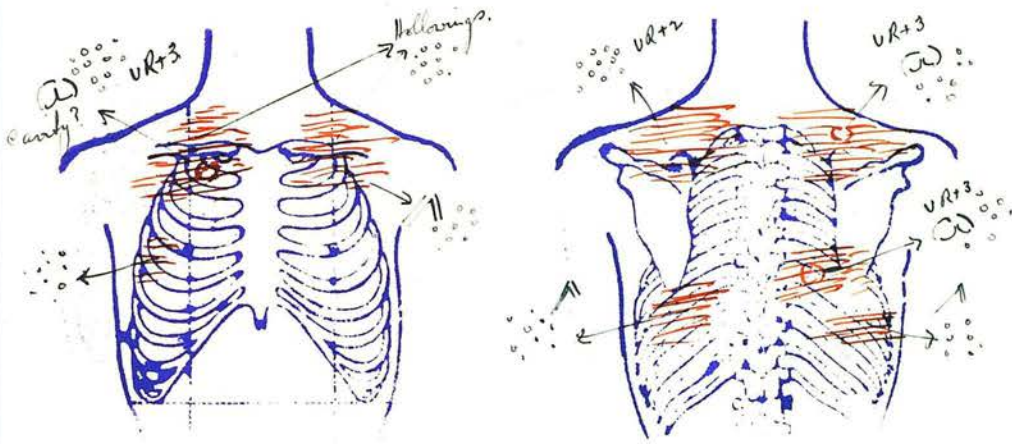
short time.

At the age of 6 years the child was attacked by Whooping Cough from which she suffered very badly, the lungs becoming markedly affected. I am assured by Dr. Stobo her medical attendant at that time, that on two occasions T.B. were found in the sputum at an interval of three months between each examination. The bacteriological report was received from the Newcastle School of Medicine on each occasion. After this the child gradually recovered, whilst to examination now all that is to be detected is a slight hollowing under the right clavicle over which area there is a little comparative dullness. At the right base behind there is dullness with diminution of the V.F. and respiratory murmurs. This is probably due to old pleuritic adhesions. Laryngoscopic examination does not reveal the slightest signs of involvement of either the true or false vocal cords.

The child looks well , has no cough, is plump and takes her food well.

CASE. No. 18.

A. J. Age 38 years. Came under my notice at Stockton 6 years ago. Ever since the age of 9 years this



patient had suffered from severe asthmatic attacks at times; for which he had used pounds of patent Asthma- powders for burning.

On examination the chest was markedly emphysematous with an expansion of one inch only 33" to 34"; the intercostal veins were markedly dilated and prominent. When last I saw him the patient was obviously in the last stages of Phthisis Pulmonalis with cavities at the right apex and in the middle lobe of the right lung and probably also at the base of the left lung. His wife informed me that he had never been very much troubled with a cough except when the asthma attacks came on which she said were terrible at times, being relieved only by the copious burning of the powders and inhalation of the fumes. He had never suffered from night sweats until just shortly before I saw him; until this time she had always imagined that the complaint

CASE. No. 18. (Continued.)

was one of pure Spasmodic Asthma.

The case was obviously one of Chronic Phth~~h~~sis associated with spasmodic asthma attacks at intervals.

CASE. No. 19.

R.A. Age $4\frac{1}{2}$ yaers. Southwick. I was **first** called about midnight to see this child in private practice in January 1915; the message was that the child was taking fits. On my arrival at the house the child seemed to have recovered perfectly from the alleged convulsions and seemed perfectly happy. The temperature was normal, the pulse 116; it had not vomited; its bowels had acted well shortly before. I could find nothing at the time to account for its condition; it had undoubtedly alarmed its mother however. The next night I was again summoned with exactly the same message and again on my arrival I found the child sitting up and apparently quite recovered. I then went carefully into the history of the supposed seizure. On both occasions the mother had been lying sleeping in bed with the child in its crib by her side, apparently quite well; suddenly she was awakened by the child's loud and difficult breathing, at the same time the little

CASE. No. 19. (Continued.)

patient was throwing its arms about and seemed in great difficulty to get its breath; the grandmother who was present on the second occasion diagnosed the condition as Asthma and said the attack passed quickly off when she took the child on her knee and warmed it beside the fire. I tried to reassure the mother that there was nothing serious, but I must confess I was completely nonplussed to find a rational cause for the condition. I asked the mother if she would bring the child to my consulting room the next day when I could thoroughly examine it. On examination in daylight next afternoon, from the dilated appearance of the veins in the upper part of the right chest, from the rigidity of the upper part of the sternum on pressure and from spinal auscultation (where bronchophony seemed to be prolonged as far as the lower part of the 1st. D.V.) I came to the conclusion that the child was probably suffering from enlargement of the mediastinal glands and that these attacks from which it had suffered were in all probability due to pressure on the trachea or large bronchi; failing this as the correct cause I must put the condition down as associated with may be enlargement of the thymus gland. As in both attacks the child was observed at the time to be lying on its back when these took place I advised the mother to train it to sleep lying on its right

OUR CHART.

DISEASE.

Highland. Pulmonary Tuberculosis

Name *P. G.*

Age *31. yrs.*

Diet

Case Book No

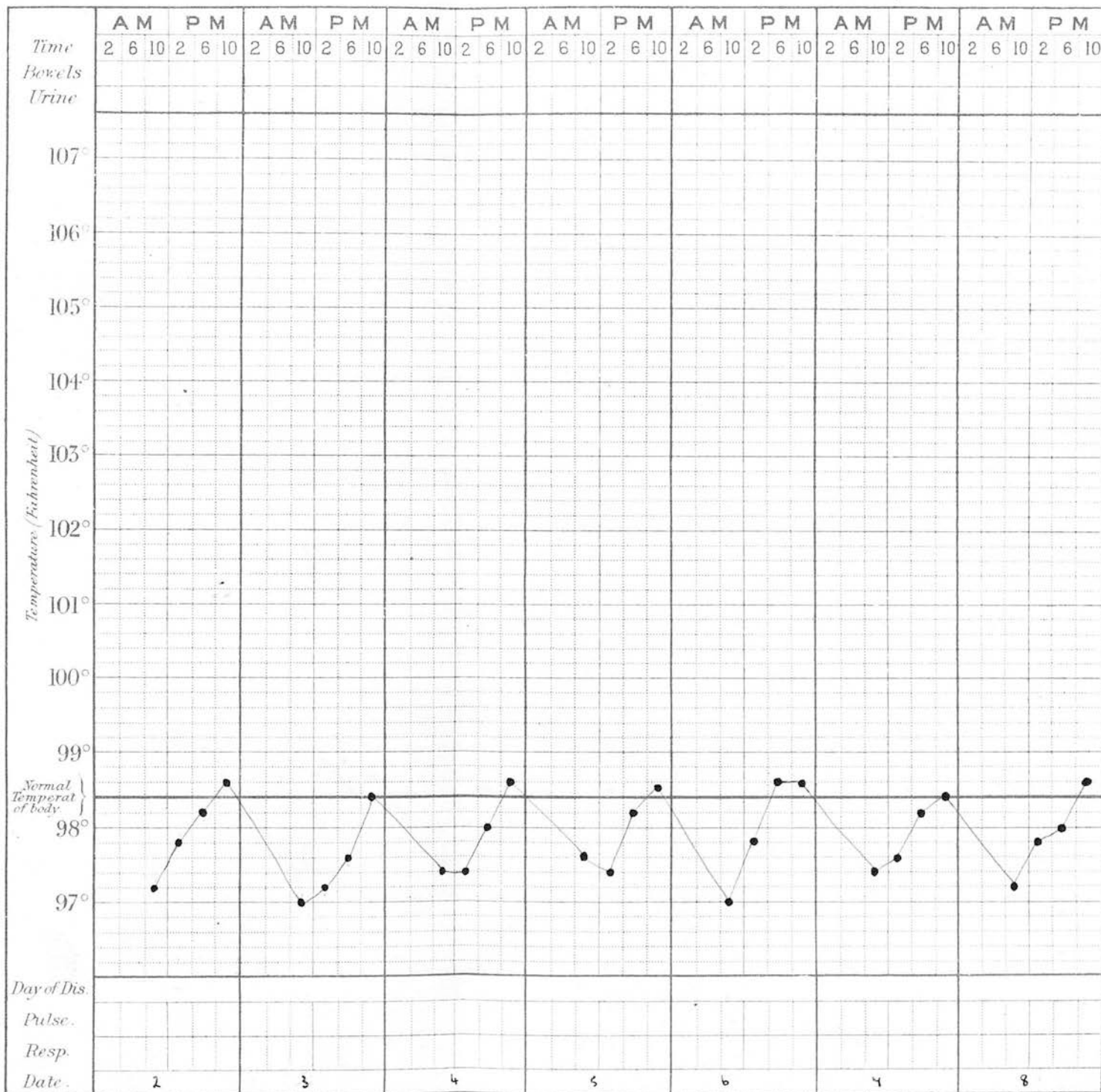
1

Notes of Case

*Ammonia.
No cough. No spitting.
Respiration slight
wt - 8 st. 2 lbs
ht - 5' - 3".*

Date of admission

Good.



CASE. No. 19. (Continued.)

thorax and with the side of its head resting on a fairly high and firm pillow. Almost six months have elapsed now since the time of writing this and my being called to see the patient I am told that the child has never had another attack since.

CASE. No. 20.

P.G. Age 31 years. Nurse. Consulted me in April 1914. Complained of loss of weight, amenorrhoea for five months, and general feeling of weakness. No cough or sputum. Occasional attacks of perspiration at nights. T.98. P.78.

On examination chest seemed well developed with moderately good and even expansion all over. Measurements 29 to 31". Height 5'-3", weight 8st. 2lbs. On physical examination nothing definite to be made out except loss of tidal expansion at the left apex behind, along with slight blurring of Krönig's area on the left side, As compared with the right. I was unable to detect any appreciable difference in the percussion notes over one apex as compared with another. No creps or alteration in the respiratory murmurs to be detected. Her muscles were soft and flabby.

Patient was advised to take her temperature every 4 hours as far as possible for a week and then to come back and report herself. During the

CASE. No. 20. (Continued.)

next week on her coming back, her chest showed a T. almost practically subnormal, ^{she} advised her to take a four or five mile walk every day, if possible taking her temperature before setting out and after coming back. Her charts are interesting especially as to the results of exercise. On the first day her walk resulted in a rise of T. of .8% above normal; on the second the rise was only .4% , on the third there was no appreciable rise at all, likewise the 4th. and 5th. On the sixth there was a rise of .9%; on this particular morning she had walked rather farther than usual and as it commenced to rain she hurried back, as a result of which she perspired very freely indeed. As I was still a little doubtful as to a definite diagnosis of active mischief I decided to inject her at an early date with a small dose of tuberculin , so I gave her an injection of .1 milligrams subcutaneously on the morning of the 17th, and told her to go to bed and that I should visit her in the evening. On my examination of her at night I found that her temperature had risen 1.1%. I was unable to detect any signs of a focal reaction however. On the next morning when I visited her I found that her T. was slightly above the normal; she informed me however that during the night her temperature had gone up still higher than it was

CASE. No. 20. (Continued.)

when I left her, ie: up to 100 and that she had felt shivery and cold.

In view of these facts I decided to treat the case as one of Incipient Active Pulmonary Tubercle. She left her situation in order to take a long holiday in the country. On my examination of her again rather more than 3 months afterwards I found her weight had increased to 8st. 10 lbs, her muscular system was very much firmer indeed, whilst she was feeling in first class health and quite another being altogether. She was advised however to continue the line of treatment for at least another six months.

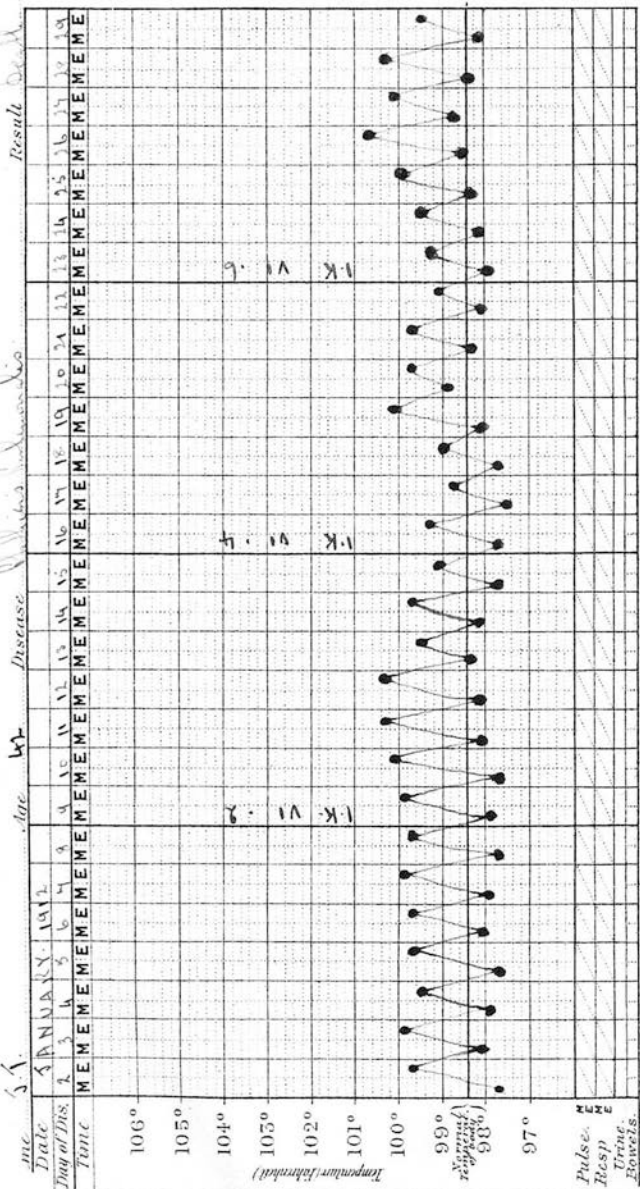
CASE. No. 21.

J. T. Age 42 years. Cashier. Consulted me in January 1912. His wife had died nine months previously from consumption. On examination patient was obviously pretty far gone in the disease. His vocal cords were evidently very much affected as he could only speak in a hoarse whisper. He was coughing a very great deal and bringing up a large amount of sputum; which on examination was found to contain a large amount of T.B. Height 5'-10", weight 9st.4 lbs, chest 34" to 35½" T 100.5. P 87.

Patient who was a very intelligent man indeed was sent home to bed and told that I should attend him at his own home. He was well acquainted with the use of the thermometer from his previous experience with his wife.

There were numerous crepitations and rhonchi all over the chest, whilst there was probably a fairly large cavity in the middle lobe of the right lung. He did not improve very much under treatment and his T. kept swinging between 97.5 and 100. My object in quoting the case is to show the effect of Spengler's Serum in which I was interested at this time and which I had decided to try on this case. The serum exerted a remarkable effect in controlling the T. as the charts will show. I have seen this effect before in other case but never so marked as in

55.



H. Silberbeck, M.D., Blackman Road, London.

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387.

CASE. No. 21. (Continued.)

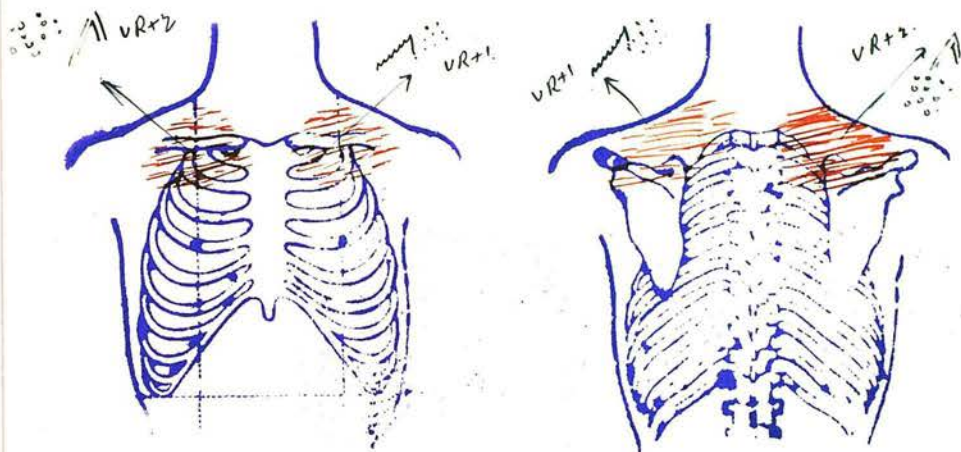
this case. There was no other effect apart from this however, to observed from the use of this Serum, as the disease still progressed rapidly; the patient dying at the end of February more from the effects of cardiac failure than from loss of lung substance or Mæxaemia, as towards the end he was in a thoroughly water-logged condition and deeply cyanosed; the feet, legs, serotum and hands being enormously swollen and dropsical.

CASE. No. 22.

W.B. Age 27 years. Hairdresser. Consulted me in July 1914. Complained of pain and vomiting immediately after food which had been going on for about five weeks. There was slight cough, and this was the only symptom suggestive of mischief in the lungs. A small diminutive type of individual, flabby muscles. Height 5'-3", weight 7st. 11lbs. Chest 30 to 31½. Flattening under the right clavicle along with diminished and lagging respiratory movement in this region. Patient looked flushed. T.100.2 P.97. and of very low tension. The history was that six weeks ago when out boating on the river and after perspiring rather freely he had caught a chill; he was laid up for five days at this time and was then able to resume work, but he had never felt the same since then.

(244)
CASE. No. 22. (Continued.)

Percussion. The right apical region was quite dull, there was also a moderate amount of dullness over



the left region but nothing like the same extent as over the right. On auscultation numerous fine crepitations were to be heard both on inspiratory and respiratory over the subclavicular region; there were also a few ^{creps} heard over the left supraclavicular area. There was thus no doubt about the diagnosis being active tuberculosis in the apices of both lungs. The patient was kept in bed for a month until the temperature had subsided; he was then sent to Stanhope Sanatorium where he remained for three months, which seemed to improve him very much indeed, for on my examination of him on his return his weight had increased to 8 st. 6 lbs; his muscles were much firmer and he was able to take his food very well indeed. I quote the case chiefly as an illustration of the gastric symptoms which were the chief cause of complaint with this patient. The vomiting used to come on almost immediately after food even of the simplest character, so much so was this, that, as he

CASE. No. 22. (Continued.)

said himself, he got no benefit from his meals at all.

CASE. No. 23

G.L. Age 19 years. Stationer's Assistant. Complained of cough, loss of appetite and being run down generally. A tall, pale and weedy looking youth. Height 5'-10", weight 9st.12lbs. Very pale and anaemic looking, and he seemed of an excitable disposition. His mother and one sister age 24 had died of P.P. On examination the chest was phthisical in type, namely, long and flat and with little expansion, 34 to 35½. Intercostal spaces retracted; veins prominent, especially in the right infraclavicular area. There was comparative dullness over both apices, more particularly over the right; the infraclavicular areas were very hollow. On auscultation there were no crepitations to be heard, but over both apices the breath sounds were feeble and interrupted in type with prolongation of expiration.

The patient insisted on my telling him the nature of his disease and was not satisfied when I gave him my diagnosis along with a fairly favourable prognosis. His subsequent history is interesting. About one week after my seeing him his sister came to ask me to go and see her brother as she thought

CASE. No. 23. (Continued.)

he had gone wrong in his mind. On my going to see him I found the patient in a most utter state of dejection; he was sitting on a chair, his head bowed down and between his hands; he was moaning and rolling from side to side. I did my best to cheer him up by telling him that I honestly thought that he would come all right again if he would only do his share.

Two days later I was again hurriedly called to see him; this time I found the patient in a state of Acute Mania requiring two persons to control him in his violent fits, which had been coming on very frequently since I had last seen him.

From the nature of his condition and as he was also markedly suicidal in his tendencies I reported the case as one of Acute Mania associated with Phthisis Pulmonalis. The patient was removed to Sedgefield Asylum where he remained for 10 months with exceedingly great benefit indeed to himself, for on his coming to see me again after his getting home I hardly knew my patient on account of the great improvement in him. He had broadened out considerably weighing now two or three ounces short of 11 stones; his face instead of being anaemic looking was quite bronzed; his muscles were firm and hard. His mental attitude seemed quite changed for the better, as he was now quite happy and exceedingly optimistic in his bearing.

CASE. No. 24.

Mrs. G. Roker. Age 83 years. Complained to me as having suffered from Chronic Bronchitis for years. Patient was very much wasted indeed when I saw her, in fact there was very little flesh on her bones at all. She was absolutely bed-fast. She expectorated a very large amount of thick nummulated sputum, here and there tinged with blood; on examination this was found to contain both T.B. and albumen. On physical examination of the chest there were great hollows above both clavicles and also below them, but in the latter respect more on the right side than the left. On auscultation the breath sounds could not be heard on account of the intensity of the râles and rhonchi heard all over the chest back and front.

Patient was evidently in the last stages of a chronic fibroid phthisis and had probably never suffered from Chronic Bronchitis per. se. at all.

She died a fortnight after I first saw her. e
The case is interesting on account of a patient of such advanced age exhibiting such physical signs.

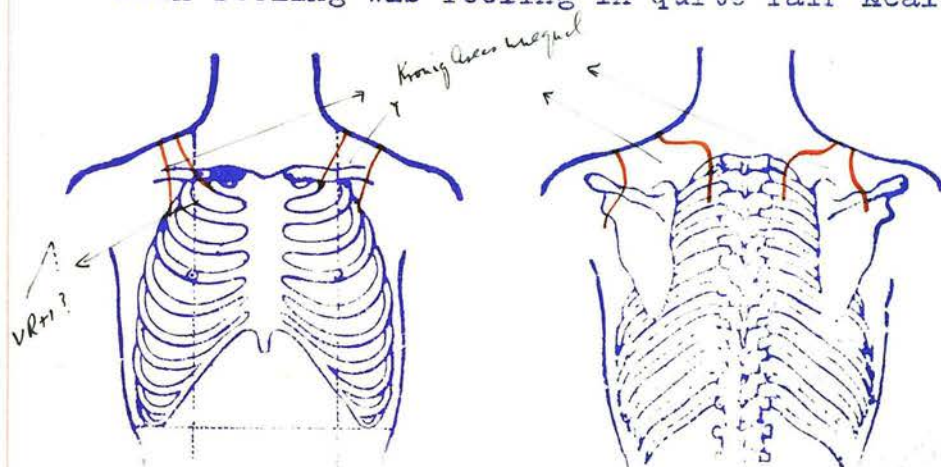
P.S. Age 66 years. Jesuit Priest. Came under my notice when in the British West Indies in 1906. Had been sent out to this region from England 18 years previously because of his suffering from consumption. The climate and life suited him so well here however that he began to pick up almost immediately after his arrival.

Patient at time of examination was a well built strong looking man and able to do the heavy work of his neighbourhood quite well and without any fatigue. The only symptom he had was the expectoration of a little sputum occasionally.

I was quite unable on my examination of him to detect any active lesion in his chest the only abnormality I could detect being a little comparative dullness over both apices in front and behind. I must confess however that at this time my powers of clinical examination of the chest were limited. I was supported in my findings by my senior colleague Dr. S. Greaves.

The interesting feature of the case was that his sputum contained T.B. and had contained them as I was assured, for certainly the past seven years. Apart from this the patient was in perfect health and did not look as if there was the slightest thing the matter with him.

A.S. Age 40 years. Fruit Merchant. Consulted me first in February 1915.. Apart from a little run down feeling was feeling in quite fair health,



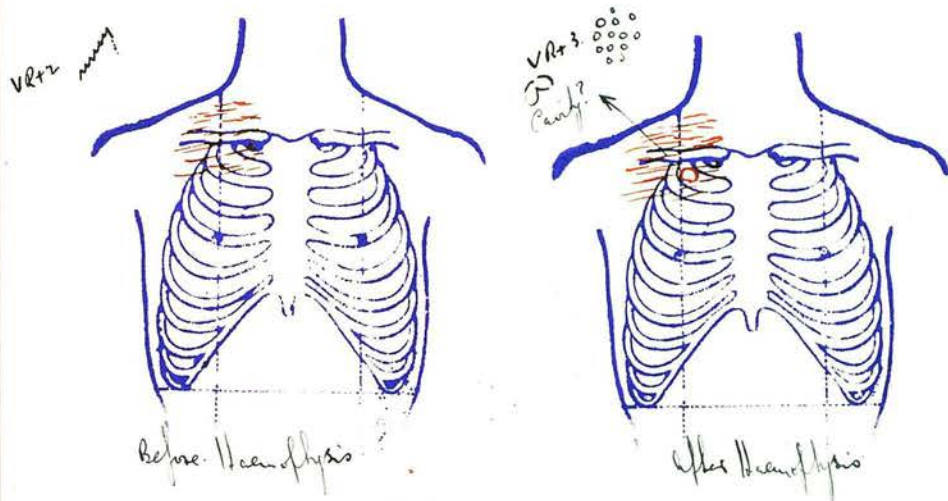
when he suddenly expectorated a mouthful of blood. On examination his chest was found to be well developed, 37 to 39½, his pectoral muscles and the muscles of his limbs however were soft and flabby. On percussion there was slight blurring of Kronig's area on the right side, this along with a rather feeble respiratory murmur over the right infraclavicular area, were the only physical signs that could be detected clinically. He was kept under observation for about a week when the haemorrhage had closed up to a large extent. On examination of his sputum then it was found to be swarming with T.B. I quote the case as illustrative of an haemoptysis being the first symptom to cause the patient to seek medical advice.

CASE. No. 27.

J.C. age 17 years. Her mother brought her to see me in June 1915. and was concerned chiefly because her daughter had had no monthly period for 15 months. Otherwise the patient was in her usual health, she had never been a strong girl yet had always been able to attend school regularly. There was no cough, no loss of weight or or any other symptom suggestive of phthisis. One sister had died six years ago from phthisis. On examination the girl was noted to be anemic her muscles inclined to be soft and flabby but this was not very marked. Ht. 5' 2" Wt. 7 stones 5lbs. Chest 28" to 30" In percussion there was marked dullness over the right apical region both in front and behind. On auscultation there was a diminished interrupted respiratory murmur only to be heard over this area. On the left side there was nothing abnormal to be made out. As apart from the amenorrhoea the child was considered to be in her usual health I hesitated to conclude at once that there was active mischief present, however the doubt was soon settled because on the third night after my seeing her I was hurriedly called by telephone to see her by her father who informed me that his daughter had just thrown up about half a pint of bright red blood. On my arrival I found that this amount was not at all exaggerated whilst now on auscul-

CASE. No. 27 (Continued)

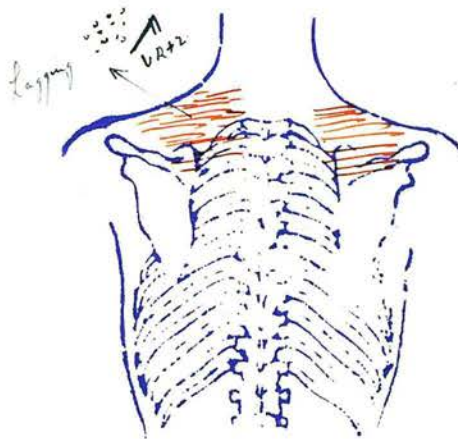
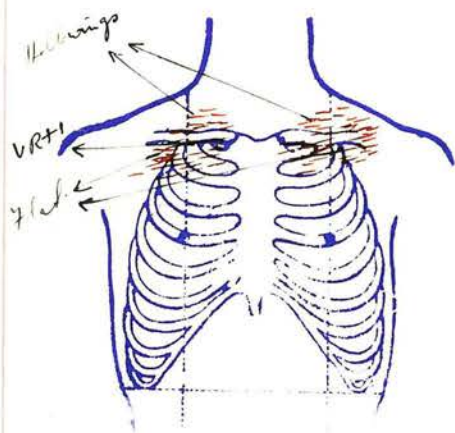
tation numerous coarse crepitant rales were to be heard all over the right apex in front and behind. ^{tense} Sedleni Muscles, and painful on right side



During my subsequent attendance on her the mother who was 55 years of age informed me that for some months herself she had been troubled with a cough, she was also quite hopeful about her daughter because she informed me she had also burst a blood vessel when she was a girl and had been ill a long time after it, although circumstances did not permit of full examination. I was allowed to make a sufficient enough examination to convince myself there were more than strong suspicions only, for assuming that the mother herself was probably an old case of hitherto quiescent tubercle which was now beginning to show signs of activity. As at the time of writing both patient are still under my care I am not yet able to complete the history of the cases.

CASE.No. 28.

A.G. age 23 years. Resident H.S. At Monkwearmouth
Hospital Jan.- June 1914. Was called by the



matron to see him one night because he had expect-
-orated about one ounce of bright blood. The
history was that he had been out of health for
about three weeks but had never complained, he
confessed that during this time, he had frequent-
-ly noticed a little blood in his handkerchief
after coughing. Previously, although never robust
he had always kept very fair health, all the time
that he was a student in E'tro he had never ail-
ed very much except a little cough at times
which he attributed to cigarette smoking.

On examination T. 100.4. P. 105. At the left
apex behind there was consolidation and a few
fine crepitations to be heard both on inspiration
and expiration; in front there were signs of old
consolidation over both apical regions. The
chest was flat in type and there was slight
curvature of the spine in the upper dorsal region

CASE. No. 28. (Continued)

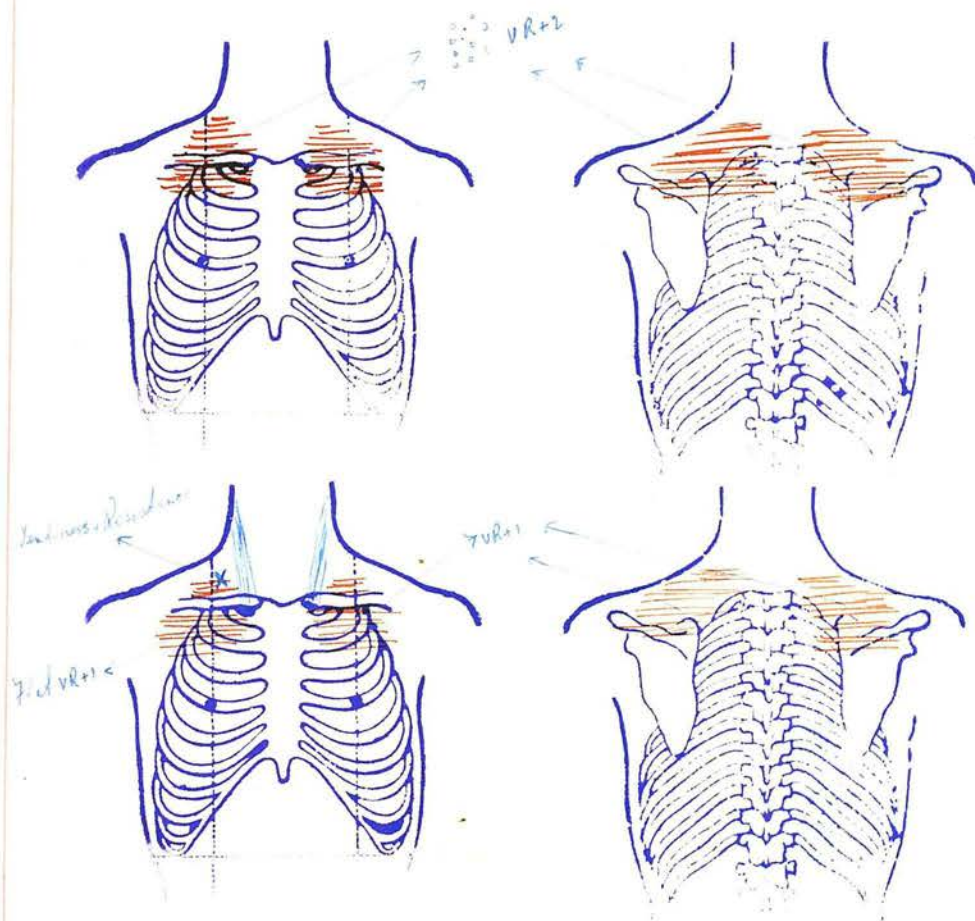
I considered the case to be one of old latent tubercle breaking down and becoming active. I gave him a good prognosis if he would carry out a rational treatment. He refused to go into a sanatorium, 8 months after I heard from him again. He stated that he was ever so much better; he had been a voyage to Japan and back as ship surgeon which had done him a world of good. I wrote to him advising him to stick to the same occupation for another 18 months at least, but as yet I have had no further news of him so I am unable to state his present condition.

CASE. No. 29.

R.L. age 21 years. Draughtsman and M.L. age 19 years Assistant librarian. The brother was rather an advanced case when I first saw him in Sept. 1913. He had active mischief associated with consolidation and crepitations over both apices, there was also impaired percussion sound and crepitations at the apex of the middle lobe of the right lung. There were numerous T.B. in the sputum and whilst his temperature was swinging between 97.2 and 100.8 I did not give his parents a very ^{hopeful} prognosis, but after six weeks treatment at home during which time his temperature subsided and became steady he

CASE. No. 29. (Continued)

was sent to Stanhope Sanatorium. He was only

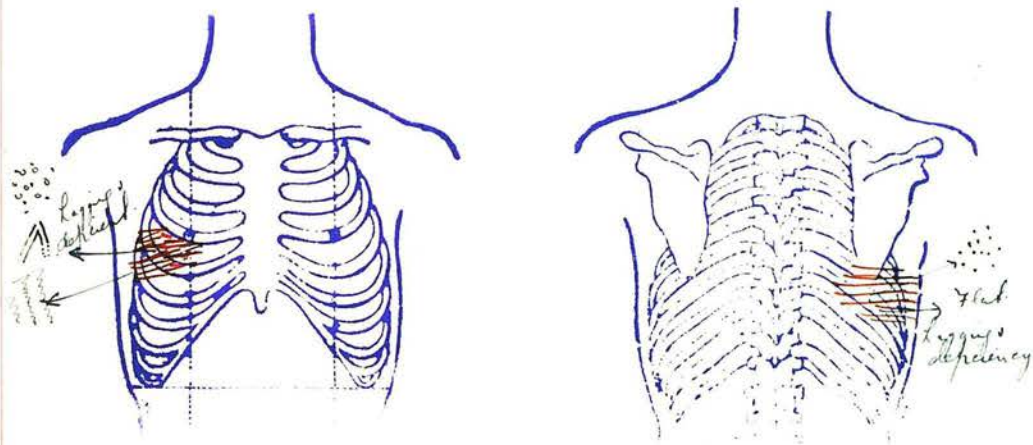


retained here for three months but his father who was an inspector on the N.E.R. got him a situation in a signal box near Stanhope where to all appearances he seemed to be improving in his health. His death came with dramatic suddenness however; about two months afterwards, he was found by one of his colleagues lying dead in a pool of blood on the floor of the signal cabin. He had evidently died from a large haemoptysis.

CASE. No. 29. (continued)

About two months after this namely in May 1914 the sister was brought by her mother to consult me. She complained of cough langnor and loss of weight. T.B. not found. The mother informed me that she like her brother had never been strong and was often troubled with colds, this one however had lasted longer than usual. Her Height was 5' 5" Weight 8stones 2 lbs. Chest 31" to 33" T. Normal 98.6 She was thin pale and extremely anaemic looking, her muscular system generally being very flabby; physical examination there was impaired sound to percussion over both apices more particularly the right along with a little flatness below the right clavicle. I was unable to detect any crepitations on auscultation. The facts which confirmed me in the opinion that the disease was active was first the tense hard resistant feeling of the (Sternocleidomastoid?) Muscles above and behind the right Clavicle, and secondly the pain felt by the patient on palpating for this sign. I never saw these phenomena so marked in any other patient, the contrast between the neck muscles and those elsewhere being particularly noticeable.

H. S. Age 43 years. Commercial Traveller. Admitted to the Monkwearmouth Hospital in July 1915 on account of his having recently sustained

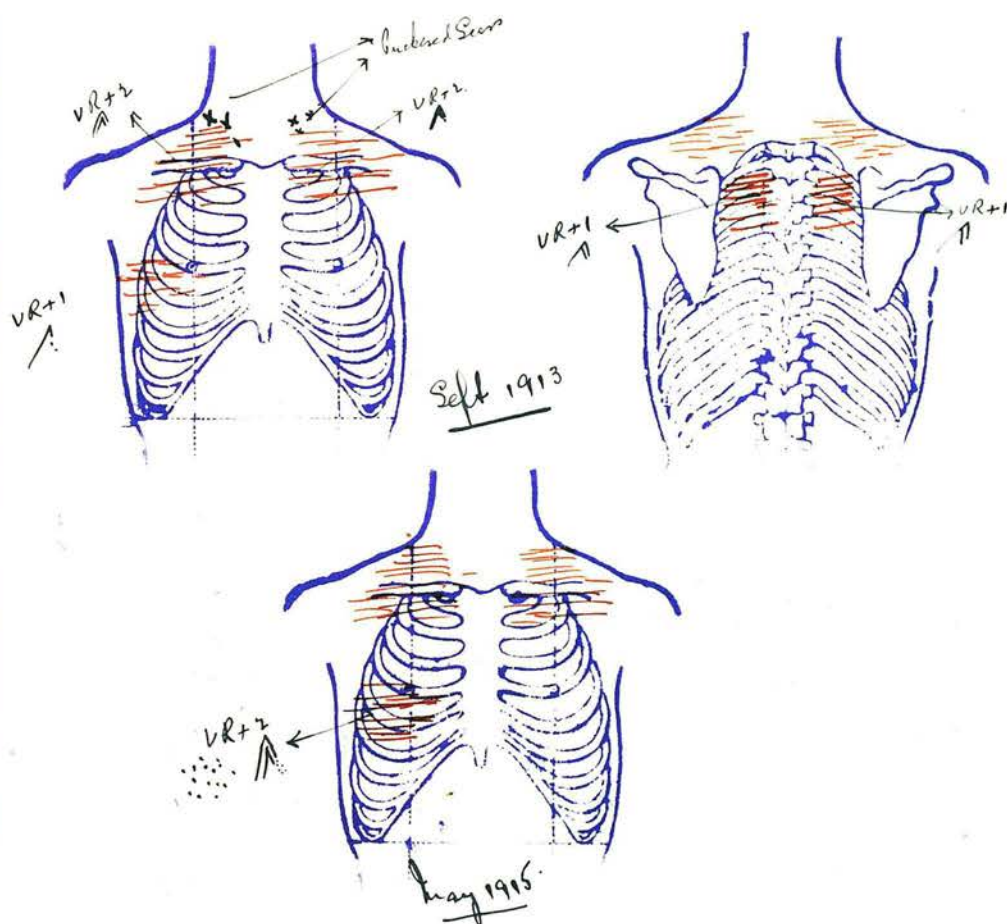


a violent attack of haematemesis or haemoptysis. The history was that this had come on suddenly whilst the patient was walking along the promenade by the sea. The blood on examination was said to be quite bright red, clear, and free from froth. It was strongly suspected that a haematemesis was the cause of the condition as it was reported that nothing abnormal was to be detected within the thorax. On examination the patient stated that he had been a life-long tea-totaller; he had suffered for some months from a cough with a little sputum at times, this had never contained blood; there was no history of a long standing dyspepsia. The patient was a thin weedy looking individual, no enlargement of the stomach or liver was to be detected and there was no area of local tenderness to be detected anywhere over the abdomen. On the

patient breathing deeply slight lagging and deficiency of the respiratory movements were to be detected over the lower part of the front of the right chest; behind there was a little flattening and diminished movement just below and to the right of the angle of the scapula. On percussion over the lower part of the right axilla it was to be noticed that there was comparative dullness as compared with the left side; on auscultation over this area a few fine crepitations were to be heard both on inspiration and expiration whilst a little further towards the front fine friction sounds were to be heard on deep breathing. Nothing abnormal could be detected in any other part of the chest, the apical regions were quite resonant on percussion. On seeing the patient again a few days after he informed us that he had had an attack of Influenza about four months ago and that he thought that he had commenced to fail in his general health since then. His sputum was still tinged with blood and was reported to contain T.B. His temperature chart showed no great elevations, he had had no attacks of perspiration; he was eating and sleeping well. The diagnosis of his condition was put down as Early Active Tubercle commencing in the Apex of the middle lobe of the right Lung.

(1261)
CASE. No. 31.

C.R. Age 27 years. Cook. Consulted me first in September 1913. She complained of palpitation on



the slightest exertion. She was a stout well made girl but exceedingly pale and anaemic looking. On examination one could see very plainly the ~~punctured~~ scars on both sides of the neck indicative of old broken down tubercular glands. There was slight puffiness under the eyes and she informed me that her feet were inclined to swell a little towards night.

On percussion there was almost absolute dullness over both apices, along with flattening below the clavicle., there was also dullness on both sides

CASE. No. 31. (Continued.)

between the shoulder blades whilstⁱⁿ the right axilla between the 5th. and 6th. ribs there was also comparative dullness as compared with the left. With the exception of feeble respiratory sounds over these areas there was nothing else to be made out on auscultation. There was no album~~in~~ in the urine. The haem~~ing~~globin was between 50 and 60 % as estimated by the blotting paper test. I was very doubtful as to whether any undet~~ected~~ active mischief might be the cause of her anaemia, but as the palpitation had only developed during the last six weeks, as her appetite was fairly good, as there was no cough nor sputum her T. was normal, and as she did not think she had lost any weight, and finally as the evidence of her chest chiefly went to show the presence of old standing quiescent tubercle, I was inclined to think that the palpitation which she complained of was not associated with any breaking down of the lesions. I tried a V.Prquat on her and sent her home to bed intending to treat her in the first place on the lines of a simple anaemia. The next day when I saw her, she had reacted most intensely to the tuberculin and she complained of great pain at the site of the inoculation; these symptoms had somewhat abated when I saw her again on the succeeding day, but she informed me that the redness and pain had increased in the previous evening to a greater

CASE. No. 31. (Continued.)

extent than ~~when~~ they were when I had seen her in the morning. This patient certainly gave the most intense V.P. reaction I ever saw. She rapidly improved in health under general hygienic treatment and the administration of iron so that she was able to resume work without any symptom of palpitation coming on with exertion as it did before.

The further history of this case is interesting. She came again to see me in May of this year viz. after an interval of one year and eight months; this time she said her health had broken down. Since she was under me before she had gone on very well indeed until she had changed her situation ~~also~~ about five months ago. Here she had had very heavy work in an underground kitchen, her bedroom also she said was in an attic which was cold and damp; it was to these factors she attributed and I think correctly so, her falling off in health. ~~She~~ She now said she suffered from palpitation ~~along~~ along with cough and a sputum also which was occasionally blood-streaked. On examination now her physical signs were as before but in addition one could now detect numerous fine crepitations just below the nipple on the right side and extending towards the axilla; these were best heard towards the end of inspiration and immediately after coughing. There was also some diminution

CASE. No. 31. (Continued.)

and lagging of the respiratory movement over this area also. I had not the slightest doubt this time that some old tubercles had broked down, Although I was unable to find any T.B. in the sputum.

As I was of the opinion that she was an suitable case for sanatorium treatment I recommen- ded her for this. She is now I believe in Wolsingham Sanatorium and the latest news I have of her is that she is improving very well indeed.

The point of interest in this case is I think the condition of the patient on the second occasion she consulted me. Had I not been aware of her previous condition, and had this been my first time of examining her I am almost certain that I should have considered the case an advanced one, whereas it was in reality only an incipient case, however long-standing one might ~~have~~ considered the lesions from a purely pathological point of view.

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CASE. No. 32.

W.R. Age 19 years. Clerk. Consulted me in July 1914. Complained of a sore throat and cough, the duration of which was about one week. Had always had very good health generally.

On examination hyperaemia of the pharynx along with marked capillary injection of the uvula was to be seen. My diagnosis was that his condition was due to excessive cigarette smoking, and on stopping this he got perfectly well in about three weeks time.

As this patient was possessed of a markedly phthisical looking chest I thought that I should have an X-Ray photo taken of his chest. The report sent back was that fairly extensive tubercular lesions were to be made out, as the result of the examination (See photo).

There was marked involvement of the postero root glands with spread from here along the bronchi in addition there was involvement of both apices more particularly the right, as on the fluorescent screen there was marked limitation of respiratory movement on the right side as compared with the left.

In spite of this however clinically, the patient does not exhibit the slightest sign of phthisis pulmonalis apart from what is seen on inspection. More for the sake of prevention than anything else I advised him to get a situation

CASE. No. 32. (Continued.)

in the open air if possible; this he has done, his firm giving him a post connected with checking the timber at the docks. He does a hard day's work, enjoys perfectly good health.

This case is probably typical of many such to which class I have frequently alluded in the text of this thesis; ^{when} when they do complain of chest affections they are often a source of difficulty from a diagnostic point of view.

CASE. No. 33.

T.D. Age 44 years. Rivetter. First came under my notice about three years ago. The history was that six months previously he had had an attack of Acute Pneumonia. He did not recover very well from this and at the end of three weeks T.B. were said to have been found in the sputum. He had remained off work since the onset of his illness up to the time, he first consulted me.

On examination the patient is seen to be an extremely well developed man, his chest measuring 40" at the nipple, weight 13st. 3 lbs, height 5'-10 $\frac{1}{2}$ ". On percussion there was slight impairment only over the right apex in front and also in the lower part of the right axilla. On auscultation there were numerous loud crackling

rales to be heard practically all over the chest back and front; these accompanied both inspiration and expiration. I have seen this patient at varying intervals ever since and have found the same auscultatory phenomena on every occasion, whilst to all appearances he is not a bit worse in his general health; although he has never been able to resume his old occupation he is able to look after ^{the} shop in his present business, of a second-hand dealer. His temperature keeps steady at the normal except for an occasional feverish attack every 6 or 8 weeks or so. There has never been any haemoptysis or attacks of perspiration. His appetite is good. There is a fair amount of sputum however, showing the much granules but no acid fast T.B. at present. For the past six weeks months he has been receiving small doses of tuberculin, once a week, but I cannot say that this has done him any good; at any rate it has made no difference to his physical signs. Four months ago I had an X-Ray plate taken (See photo) but it does not show lesions anything like corresponding to what one would expect, judging from the auscultation signs. There is certainly a denser hilus shadow than normal with strands spreading out into the lung from this region in all directions.

I am not yet certain as to the exact nature of the lesions but think that probably the

CASE. No. 33. (Continued.)

primary seat of the disease is in the glands at the root of the lung, with extension along the tubes and lymphatics from here into the substance of the lungs themselves.

CASE. No. 34.

L.M. Age $2\frac{1}{2}$ years. Residing in Monkswearmouth. Father away on active service. Was first called to see this child in January 1915, when my diagnosis was that it was a case of Acute Bronchitis. The child was pale anaemic, also slightly hydrocephalic, the ant. fontanelle being still open; the veins over the temples and scalp were very prominent as were also the intercostal veins. The child in spite of every care did not seem to show any signs of recovery yet I was unable to find any trace of an empyema on either side. Towards the end of the 14th. day symptoms suggestive of Bacterial Meningitis made their appearance; the child now lay perfectly still with the head slightly retracted, there was internal strabismus of the right eye along with slight ptosis of the upper lid; the abdomen was retracted and there was marked 'tache cerebrale' phenomenon on scratching the skin over the abdomen; breathing instead of being fairly rapid as formerly was now very shallow and feeble.

CASE. No. 34. (Continued.)

At this time when looking round the room I noticed for the first time a temperature chart pinned to the wall. On questioning the mother regarding this she told me it was her own. Four months previously she had returned from Wolsingham Sanatorium and was now attending twice weekly at the Borough Tuberculosis Dispensary where she was receiving tuberculin treatment and it was in connection with this that she was required to keep a record of her temperature. I did not examine her but she told me that she still coughed a great deal and occasionally brought up some sputum. Obviously she was still in an active stage of the disease and I was satisfied inasmuch as that I had discovered the source of infection in the child, who died on the 16th. day of its illness from evidently an Acute Generalised Tuberculosis.

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EXTRACTS REGARDING DEATHS FROM
PULMONARY TUBERCULOSIS FROM REPORT OF
REGISTRAR GENERAL. 1913. P184.

AGE.	MALE.	FEMALES..
UNDER 5 yrs	437	365
5—9 yrs	184	248
10—14 yrs	262	631
15—19 yrs	995	1438
20—24 yrs	1752	1737
ALL AGES. TOTAL	19056	14363.

TOTAL D.R. FROM PHTHISIS PULMONALIS = 1004 PER 1000000
LIVING = 7.3% OF TOTAL MORTALITY AND IT FORMS 75% OF ALL
DEATHS FROM ALL TUBERCULOSIS WHICH FORMS 9.8% OF TOTAL
MORTALITY FROM ALL CAUSES. (P. XLIV).

ANALYSIS OF VON PIQUET REACTIONS

No. of CASES.			AGE PERIOD.		POSITIVE		NEGATIVE		PERCENTAGE		
M.	F.	M, F.			M.	F.	M.	F.	M.	F.	M & F.
21	20	41	5 to 9 yrs.		12	14	9	6	57	70	63.4
19	8	27	10 to 19 yrs		10	5	9	3	52.6	62.5	55.5
42	26	68	20 yrs +		36	19	6	7	86	73	81
82	54	136	ALL AGES.		58	38	24	16	70.7	70.4	70.6

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* Excluding the number of cases in this class we have small & give reliable results.

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